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Pathological Anatomy.

ILLUSTRATIONS

OF THE

ELEMENTARY FORMS OF DISEASE.

BY

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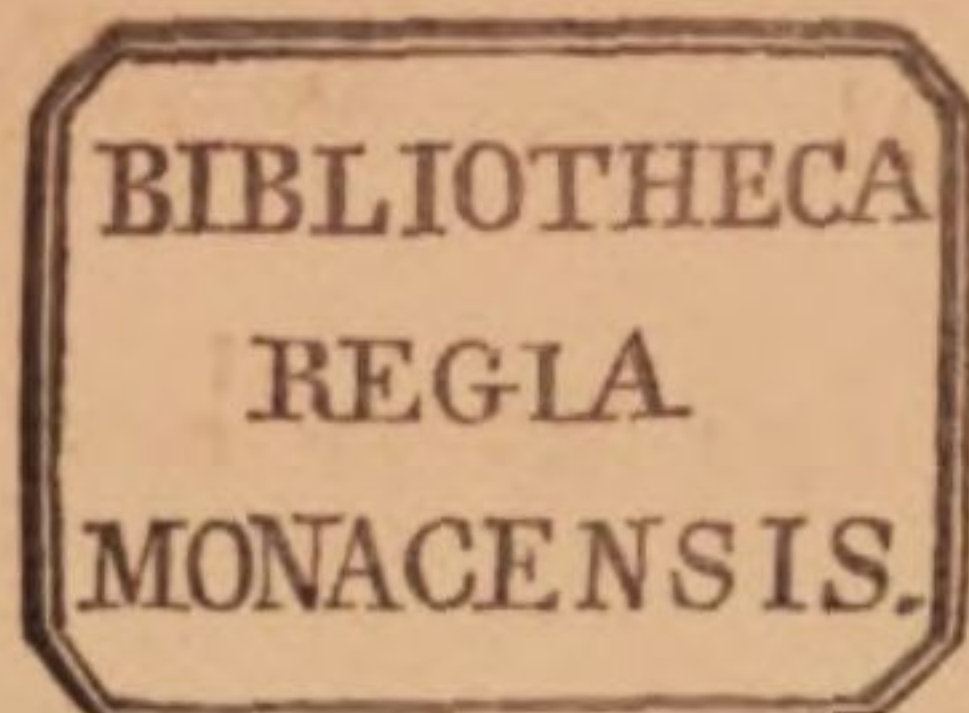
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1838.

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*Carswell
Pathological
Anatomy*

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TO

JAMES JEFFRAY, M.D.

PROFESSOR OF ANATOMY AND PHYSIOLOGY IN THE UNIVERSITY OF
GLASGOW, ETC. ETC.

MY DEAR SIR,

THE approbation with which you received my first attempts to represent, by coloured delineations, the healthy and diseased appearances of the human body, while attending your Lectures, and the encouragement and assistance which you afterwards afforded me in the prosecution of the subject, lead me to consider the publication of this work a befitting occasion on which to express my gratitude, and to acknowledge the influence you thus exercised in directing my attention in a special manner to the study of Pathological Anatomy. That this work, which embraces but a part of the subjects which derive illustration from a department of medicine to the cultivation of which I have devoted much time and labour, may also meet with your approbation, and be the means of extending our knowledge of diseases, is the desire,

MY DEAR SIR,

Of your sincere and grateful servant,

ROBERT CARSWELL.

Berners-Street,

Oxford-Street, London,

15th December, 1837.

INFLAMMATION.

OF all the diseases to which the human body is subject, inflammation is by far the most frequent ; and, occurring in all the organs and in almost all the tissues ; preceding, accompanying, or following a great many other important diseases ; the precursor of various local lesions, and numerous solid and fluid products ; and the necessary condition or means by which most injuries and all solutions of continuity are repaired, an accurate and extensive acquaintance with the phenomena which characterise its existence, constitutes the most essential part of the elementary education of the student in pathology. It is, however, the local phenomena only of inflammation which I propose to consider in this place, and more especially those by means of which its presence is to be detected after death. But before entering upon the consideration of these, I shall premise a few general observations on the nature of this pathological state.

Nature of Inflammation.—Notwithstanding the numerous experiments that have been instituted to determine the nature of inflammation, much diversity of opinion still prevails, more especially regarding the state of the capillaries in the affected part. That such should be the case is not surprising when we consider that the properties of these vessels, and the part which they perform in the circulation, are points on which physiologists are far from being agreed.

To determine the changes which take place in the capillary vessels, and in the circulation of the blood through them, in inflammation, various chemical, mechanical, and physical agents, which excite the sensibility and contractility, have been applied to the transparent parts of animals exposed to view under the microscope. The results of the experiments made under these circumstances are, in many respects, extremely unsatisfactory, and, as regards the state of the capillaries, often contradictory ; these vessels being represented by some as manifesting no change of capacity, by others as contracting or dilating on the application of the same agents. The same may, in some measure, be said of the motion of the blood, which, although generally represented as accelerated when the capillaries are contracted, and retarded when they are dilated, is also said to undergo both changes in the opposite states of these vessels. If, however, we carefully examine in detail the numerous experiments which have been made on this subject by Thomson, Wilson Philip, Hastings, Kaltenbrunner,

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Wedemeyer, and Gendrin, and separate the effects of chemical or other agents which are known to modify the tissues and the blood in a manner very different from that of a mere stimulus, from those produced by this latter cause, we shall find that the results have almost always been similar in kind under similar circumstances, and in perfect accordance with the known laws of the vital manifestations of sensibility and contractility,—properties, the sensible modifications of which, if not the most important, are the first that occur in the series of changes which constitute the state of inflammation. Equally important, because constant changes, are also those which take place in the temperature of an inflamed part and in the vital and physical conditions of the blood, and, as immediate consequences of these, in the functions of secretion, absorption, and nutrition.

Considered in a general point of view, the changes to which I have just alluded as constant conditions of inflammation, may be considered either in the order of their succession, or as distinct groups characterising a difference in the stage or period of the disease. I shall adopt the latter method, and shall consider inflammation as a disease the essential phenomena of which present themselves in two successive stages or periods, each of which is referable to opposite conditions of the physiological properties or functions of the affected part. Although these opposite conditions are sufficiently characterised by the changes which take place in the temperature of the inflamed part, and in the functions of circulation, secretion, and absorption, they are more especially so in those which occur in the sensibility and contractility. The changes induced in these two vital properties are, as I have already stated, the first that are observed in inflammation from the influence of stimuli, such as mechanical irritants. The first effect of mechanical irritation is an increase of the sensibility, soon amounting to pain, even in those tissues which, under ordinary circumstances, possess this property in a very low degree; and subsequently, or at the same moment, contraction, or an increased development of the contractile property, if not of the capillaries, of the arteries with which they immediately communicate. After a certain time, varying with the violence of the exciting cause, the sensibility diminishes or ceases to be manifested, for pain is no longer the consequence of the same cause. The contractility undergoes a similar change; the minute arteries cease to contract when stimulated, and they, as well as the capillaries and minute veins, are permanently dilated and distended with blood. That these two opposite states of the sensibility and contractility exist in inflammation, and occur in the order in which I have stated, might, independently of the evidence of the facts themselves, have been believed and understood; for they are but an exaggeration of the law of vital action, or of the physiological conditions of all parts endowed with these properties, a state of activity and repose, of increase and diminution, being the opposite and invariable consecutive conditions of both. It is hardly necessary to observe that these properties, both pathologically and physiologically considered, admit of these opposite conditions in so far only as the exciting cause acts in the strict sense of a stimulus; for if the agent to the influence of which they are subjected is of such a kind as to destroy, or, as is commonly said, to exhaust, instead of an increase there is at once produced a diminution, cessation, or extinction of both, and, consequently, of their visible signs. Such a state cannot, therefore, be regarded as a state of inflammation; for the first, or active, conditions of the disease have not existed. It is, however, a state

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which is frequently produced in experiments made to ascertain the effects of stimuli on the capillary circulation in inflammation, and then becomes an interesting illustration of the fact just stated; for although congestion takes place in the part thus debilitated, it is soon followed by the active phenomena of inflammation, or those which characterise its first stage, and a more rapid and extensive formation than usual of those of the second.

Next in succession and importance to these opposite states of the sensibility and contractility, are the changes which take place in the hydraulic conditions of the blood. In the first stage of inflammation, the circulation is accelerated, and a greater quantity of blood than natural passes into the capillaries; in the second stage, the circulation becomes impeded, and the blood which distends the capillaries ceases at last to circulate. That these changes in the circulation of the blood are the consequences of the difference in capacity of the capillaries in the first and second stages of inflammation cannot be satisfactorily ascertained; but what is of greater importance is the relation which exists between them and the primary lesions of the sensibility and contractility, as they correspond to the two opposite states of these properties, viz. in increased and diminished circulation, which implies opposite states of the agents which effect or regulate the transmission of the blood in the capillary vessels. There can be no doubt that other causes than these modify the circulation of the blood in the capillaries, in inflammation, independently of any increase or diminution of the propelling power. The diminished capacity of the smaller arteries in the first stage of the disease, and the dilatation which they present in the second, certainly offer, on hydraulic principles, a rational explanation of the increased and retarded circulation of the blood in each stage respectively. An increased vital affinity between the blood and the capillaries has also been supposed as a probable cause of accelerated circulation, and the subsequent accumulation of the blood, such as is observed to take place in erectile tissues during the state of turgescence; and changes in the blood itself, more especially its coagulation, which, in the second stage, must mechanically impede the circulation, and increase in degree and extent the local congestion. All these modifying causes of the capillary circulation must, however, be regarded as secondary in importance to those to the agency of which I have already ascribed the two essential changes which occur in it in the first and second stages of inflammation.

In further corroboration of this view of the nature of inflammation, I shall briefly notice the other changes which take place in this disease, viz. in the vital and physical conditions of the blood, and in those of the temperature, secretion, and absorption. During the first stage, the vital properties of the blood undergo a manifest increase. A greater quantity of fibrine is formed, the plastic property of which is increased; for, besides its rapid organisation under favourable circumstances, it retains, when separated from the other constituents of the blood, its fluidity for a longer period, and contracts more firmly, than in the natural state. The unwonted vigour of the circulation in general, the increased temperature of the whole body, and the resistance opposed to the means employed in inflammation to weaken the vital powers, may also be regarded as connected with this state of the blood, or as inordinate effects of its vital agency through the medium of the nervous system. In this stage, too, the temperature is variously and greatly increased; the function of secretion is also

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for a time augmented; in glandular organs, however, only at the commencement; in serous tissues for a much longer period, and to a much greater degree. Absorption, if not increased, manifests its activity by the speedy effects of poisons locally employed in this stage.

During the second stage of inflammation, changes the reverse of those just described occur in rapid succession. The blood ceases to circulate, coagulates, and assumes a dark colour; the temperature sinks, and secretion, absorption, and nutrition are finally interrupted. If those conditions of the affected part are maintained for a certain time, new products are formed, or other diseased states are produced, as softening, suppuration, ulceration, and mortification, which are therefore denominated *terminations* of inflammation, and constitute separate subjects of investigation.

From this general view of inflammation, the facts sufficiently prove that it is not a disease the phenomena of which can be explained by a reference to the exclusive doctrines of *increased* or *diminished action* of the vessels. It is obviously a compound of both, and not merely of the vessels of the inflamed part, but primarily and essentially of the function of innervation also, of the vital properties of the blood, and, consequently, of organic composition. This view of inflammation is likewise in accordance with the effects of the remedies employed for the cure of the disease. Like the disease itself, the remedies by which it is successfully combated possess opposite properties or produce opposite effects. They excite or depress, increase or diminish the vital powers either of the whole body or of a part, and are beneficial in so far only as they are employed in opposite conditions of the disease, that is to say, in each of the two stages respectively of which it is composed.

A difference in the degree or duration of inflammation; in some of its local characters, or of the general symptoms which accompany its progress; in the constitution of the individual in which it occurs; in the manner in which it terminates; in the nature of the products to which it gives rise, as well as the existence of other diseases with which it may be associated as cause or effect; has led to the use of terms expressive of the existence of various kinds of inflammation. Such are the terms:—adhesive, suppurative, ulcerative and gangrenous inflammation; of acute and chronic, active and passive; erythematous, exanthematous, phlegmonous; rheumatic, gouty, syphilitic, scrofulous, cancerous, &c. But whatever practical importance these distinctions of inflammation may possess, it must be borne in mind that they do so only in reference to the special condition to which they are applied; that inflammation *per se* must always be considered as the same in its nature, as consisting of the same modifications of vital property and function; and that, however much the solid and fluid products, the local and general symptoms may vary in number or nature, there are only a few which derive their existence immediately from this pathological state, the existence of all the others being dependent on other pathological states, each requiring its own conditions, and having, like the former, its specific or distinctive characters.

Physical characters of Inflammation.—I shall now proceed to describe the physical, or, as they are sometimes called, anatomical characters of inflammation, excluding those of a physiological character already noticed, and consequently those also derived from the presence of coagulable lymph, pus, ulceration, and mortification, our chief object being to ascertain the existence of the disease by means of those changes which

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are peculiar to itself, or which distinguish it from other diseases which it resembles. The physical characters of inflammation consist in modifications in the natural colour and vascularity, in the consistence and bulk of the inflamed part.

Increased *redness* and *vascularity* constitute the most conspicuous of the physical characters of inflammation. They vary much in degree, and are not equally present in all tissues. They occur also under several forms, which, as they indicate differences in the seat, mode of extension, stage or degree of inflammation, merit the special attention of the pathologist. They may be considered as constituting what may be called the *primary* and *secondary* forms of inflammation. The *primary forms* are as follow:—the *ramiform*, *capilliform*, *uniform*, *punctiform*, and *maculiform*. The first and second of these, as the terms imply, have their seat in the small arteries and veins, and in the capillaries respectively; the third and fourth are produced by capillary injection, the uniform red colour which accompanies the former being the consequence of an accumulation of blood in the entire capillary system of a part; the minute dotted or punctiform redness of the latter arising in the peculiar structure of the part, as in the villosities of mucous membranes, when the seat of inflammation, apart from the mucous tissue itself. The maculiform has also its seat in the capillary vessels, is sometimes produced by the accumulation of blood being greater in some points than others, but much more frequently by rupture of these vessels and extravasation, and hence may be called the *hæmorrhagic* form of inflammation. Exclusive of the influence of various modifying causes, but more especially of the quantity and quality of the blood, the several degrees of inflammation are expressed by each of these forms in the order in which they are enumerated, the ramiform indicating the least, the maculiform the greatest degree.

Uniform redness is best seen in dense vascular tissues, such as the skin, where it always occurs as the first physical sign of inflammation. Vascularity, on the contrary, is seldom conspicuous; and hence, when describing cutaneous eruptions, as the exanthemata, we employ the term redness rather than that of vascularity, to indicate the kind and degree of the inflammation which these eruptions present. Uniform redness is also observed in inflamed mucous membranes, but it is always preceded by the capilliform or punctiform injection. In these membranes also, the ramiform is remarkably conspicuous, and the maculiform or hæmorrhagic more frequent than in any other tissue. All these forms indeed, varying in degree, are met with combined in the mucous membranes, more especially of the digestive organs, the capilliform and punctiform being the first that appear, and the most characteristic of inflammation. In parenchymatous organs inflammatory redness is strongly marked; but from the complex structure of these organs, and the great quantity of blood with which they become impregnated during life or after death from a variety of causes, it is often extremely difficult to ascertain how far the redness depends on this circumstance or on capillary injection. In organs naturally of a pale colour, as the brain and nerves, lymphatic glands, testes, mammæ, &c., redness and the causes of it are sufficiently obvious. It depends on the uniform capillary injection of the first or second stage of inflammation, and is succeeded by the punctiform and hæmorrhagic, which indicate a progressive increase in the disease. In all the serous membranes uniform redness is produced in the same manner, the capilliform and punctiform injection appearing first, and giving rise, as the inflammation proceeds, to the formation of striæ or maculæ, which are distinguished from the former by their darker red colour. The ramiform injection never occurs in these membranes,

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this appearance, when it accompanies the other physical characters of inflammation, having its seat in the subjacent cellular tissue. It has, indeed, always appeared to me that even the capilliform injection of these membranes is in great measure produced by the penetration of the blood into their softened substance by the *vis à tergo*. I have certainly never been able to discover bloodvessels in the arachnoid, in inflammation of the pia mater, except where it lies in contact with this membrane.

In all membranous tissues the several forms of inflammatory redness may be fictitious, that is to say, the red colour which these tissues frequently present may be owing to the blood accumulated in the subjacent cellular tissue, the colour of which is transmitted through them according as they possess different degrees of transparency. With regard to the degree or intensity of the redness of inflamed tissues, it is hardly necessary to observe that it must be estimated by comparison with the natural colour of the tissues in the healthy state. That there may be no deception with regard to the degree and nature of the red colour and vascularity of parts after death, it is also of great importance that they be examined immediately they are exposed to view, as, under the influence of the air, those which are almost pale become reddened, or, if slightly red, become much redder in the course of a very few hours. From this cause membranes in which few or no bloodvessels are at first observed by the naked eye become vascular, and venous and mechanical congestion assumes the arterial aspect of inflammation.

The *secondary forms* which inflammatory redness presents constitute an important feature among the other physical characters of inflammation, more especially in the cutaneous and mucous tissues. In the former they constitute the distinctive local characters of the exanthemata, and mark peculiarities in several of the papular, tubercular, vesicular, pustular, and scaly eruptions; in the latter they serve to distinguish the circumscribed and diffuse inflammations to which these membranes are subject, as well as the anatomical element in which they are seated, as the villous, mucous, and follicular structures. Thus in erythema, the most simple of the acute cutaneous affections, the secondary forms of redness occur in diffuse continuous patches of various extent, of a round, oval, or irregular form. The varieties which it presents are derived from the external appearances of the patches,—appearances which are produced by the degree of congestion of particular points, or of the whole of the inflamed portion of the cutis. When, for example, the redness is uniform, as in intertrigo, or when heat or a blister has been applied to the skin for a certain time, we have what is called erythema simplex. But when the cutis forms projections of various sizes, rendering the surface of the patches unequal or swollen, we have produced the varieties of this affection called erythema papulatum, tuberculatum, nodosum, &c. Inflammatory redness furnishes no distinction between erysipelas, in its early stage at least, and erythema simplex, except in degree and extent; and this distinction is important, for it is owing to these two circumstances that the redness does not disappear on pressure when erysipelas is at its height, the blood being retained in the capillaries for the reasons already stated. In rubeola and scarlatina the degree and form of the redness are in most cases sufficiently characteristic of each. In both it appears in points; but while these, in the former, give rise by their union to semilunar or crescentic patches, in the latter they produce irregular patches of much larger extent. Nor is the red colour so bright in rubeola as in scarlatina, probably owing to the simultaneous inflammatory congestion of the respiratory mucous membrane in the former disease. The

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redness in roseola, a disease which has often been confounded with rubeola and scarlatina, assumes an annular or more rounded form than that of either of these two diseases; the patches are more circumscribed, larger than those of rubeola, and smaller than those of scarlatina. Of the six species of urticaria described by Willan, in three only does redness constitute one of their physical characters, viz. in urticaria febrilis, conferta, and tuberosa. In these it forms patches, streaks or bands, having the shape of wheels, irregular stripes or projections of various dimensions. The redness is not continuous; it is eccentric; that is to say, it surrounds an elevation of the cutis, which either preserves its natural colour, or is less red than that which forms its border, and has a glossy aspect from the serosity with which it is infiltrated.

The secondary forms of redness in inflamed mucous membranes differ considerably, and are important, as they often indicate, as I have stated, the precise seat of the inflammation. In the villous mucous membranes, as the digestive, the redness, at first punctiform and afterwards uniform, occurs in circumscribed patches; in the follicles it takes the form of these bodies, or when confined to their orifices, their basis, or both, appearing in dots, rings, or stellæ,—forms of inflammatory redness which distinguish it from every other. In the non-villous mucous membranes, or when confined to the mucous tissue, the redness occurs in diffuse patches which have no definite form.

Besides the changes of colour and vascularity which I have noticed thus generally, as accompanying the acute or sthenic states of inflammation, there are others to which I may make some allusion in this place, and which are observed to accompany the chronic or asthenic states of the disease, such as various shades of purple, brown, and black. These changes of colour take place when the circulation has been retarded for a considerable time, or when it has ceased in the capillaries, and more especially when the inflammation has not terminated in suppuration or the effusion of coagulable lymph. The different shades of brown and black which originate in this state of the capillary circulation are not observed in all the tissues in the same degree; they are also more frequent in some than in others, and like the red colour of acute inflammation, present considerable variety in the forms which they assume. When the circulation has only been retarded, the colour is brown or perhaps only of a high venous tint; and where there has been complete stagnation of the blood, the colour is black. The first degree of discolouration is seen in chronic inflammation of the eye, of the mucous membrane of the air-passages, and of the intestinal canal. In the eye it has almost always a ramiform aspect; in the intestinal canal it is more frequently punctiform, more particularly when seated in the villousities, producing a grey or slate colour, called by French pathologists *couleur ardoisée*. Sometimes, also, it occupies the orifices of the follicles, and appears in grey or dark-blue points; or having its seat in the mouths or basis of these bodies, in little rings or circles. In the mucous membrane of the air-passages it is much less common than in that of the stomach and intestines, is more uniform and much less marked; and in the mucous membrane of the urinary and generative organs it is often great in degree and considerable in extent. It is not frequently met with in serous membranes unless when complicated with other diseases; and in parenchymatous organs it is most conspicuous in the lungs, in which it exists alone or accompanies other diseases, as chronic hepatization and tubercles. It is important, however, to remember that similar discolourations are met with in tissues

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without being preceded by chronic inflammation, and which I have described under the head of Spurious Melanosis.

The next and most important physical character of acute inflammation is a diminution of *consistence*, or, more correctly, of *cohesion*, of the organic elements of the affected part. This change commences in the first stage of inflammation, and may proceed to such a degree in the second, as to render even the bones soft and fragile, and convert all the tissues into a mere pulp. It appears to affect the uniting cellular element more than any other, of tissues and organs, and to do so in proportion to the degree of the inflammation by which it has been preceded. It is on this account that mucous and serous membranes which cannot, in the healthy state, be separated from the parts they cover but by dissection, are easily removed, or peel off when pulled; that muscles are detached by merely running the finger between them; that the muscular coat of the intestines is unsheathed as it were from its serous and mucous coverings, and the bloodvessels withdrawn from the substance of organs. A diminution of cohesion is thus made known in many cases of inflammation where redness and vascularity have disappeared, or mark but faintly the degree of alteration which the disease has effected in the process of nutrition.

In parenchymatous or spongy organs, and in all the tissues the structure of which favours the retention of the effused fluids, a state of solidification, called *hepatization* in the lungs, is produced; but although they then feel harder than natural when compressed, the diminution of cohesion which has taken place between their anatomical elements is rendered conspicuous by the facility with which they are penetrated, broken down, or crushed. The necessity and importance of recognising this physical character of inflammation must, therefore, be obvious, as it not only enables us to detect the existence and appreciate the degree of this pathological state, but also to recognise the first and most simple perceptible organic alteration to which it gives rise.

An opposite condition, a state of *induration*, is a frequent consequence, or at least often accompanies inflammation when *chronic*. It differs from the solidification in acute inflammation in this, that there is at the same time increased cohesion of the anatomical elements of the affected part. Tissues, instead of being more easily separated as in the former state, are more firmly united, or so often confounded together as to have lost their distinctive characters; and soft, spongy, or cellular organs frequently acquire a very great degree of density and tenacity. It is obviously the consequence of the organization of the coagulable lymph effused in the first or second stages of acute inflammation, either into the interstitial cellular structure of organs, or into the tissues themselves, in the previous state of softening which I have described. It is always accompanied by an increase of thickness or bulk, by the grey discolouration of chronic inflammation, and by a greater or less degree of opacity of all membranous tissues.

An increase of *bulk*, thickness, swelling, or tumour, always accompanies acute inflammation. As it depends on the accumulation of blood which takes place in the capillaries, and the effusion of serosity and coagulable lymph, when retained, it must vary in degree with the quantity of these fluids, and present considerable difference of form and extent in different organs and tissues. It is always most conspicuous when the inflammation is circumscribed, as in furunculus, in papular and tuberculous affections.

NOTICE.

THE great difficulty, and frequently the impossibility, of comprehending even the best descriptions of the physical or anatomical characters of diseases, without the aid of coloured delineations, induced me to undertake the publication of the present work; and that it might be made as accessible as possible to the Profession generally, as well as to diminish in some degree the labour and expenses attendant on such an undertaking, to publish it in fasciculi.

As originally announced, I have comprehended the Illustrations of the Elementary Forms of Disease in twelve fasciculi. There are, however, some subjects, interesting in themselves, and well adapted for illustration by means of coloured representations, viz. Calculi, Entozoa, and Monstrosities. These I propose to publish when a second edition of the work shall be required. They will form an appendix, and, along with any additions or alterations that may be made in the letter-press of the second edition, may be obtained by subscribers and others in possession of the first. I may observe that the order in which the fasciculi have been published is the reverse of that in which they should be arranged when bound. It was unavoidably adopted, and may be easily remedied on referring to the Table of Contents, which, for the reason assigned, must also serve as an index to the separate subjects.

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of the skin, in the inflamed papillæ of the tongue in scarlatina, and the villous and follicular structures of mucous membranes. It is equally marked in inflammation of the mamma, testis, and kidney, and even in the lung, which, in consequence of the degree of this change in its bulk, sometimes receives the impression of the ribs against which it has been pressed. It is, however, an alteration which acquires importance rather from the effects to which it gives rise, as compression and occlusion, than from its value as a physical character of acute inflammation.

Diagnostic Characters of Inflammation.—Although the existence of inflammation is, in general, sufficiently characterised by its physical characters, it may be, and frequently is confounded with other local accumulations of blood, produced during life or after death. A *mechanical* obstacle to the return of the venous blood; the *depending position* of organs, or *gravitation*; and the transmission of the colouring matter of the blood to neighbouring tissues by *imbibition*, are circumstances under which the local accumulations of blood, redness, and vascularity take place, which require to be distinguished from similar changes produced by inflammation. The differential characters of inflammation and local congestion are founded on certain differences in the physical characters of each, and in the circumstances under which they respectively occur. Although similar forms of redness and vascularity are produced in both, it is perhaps only in mucous membranes that a difficulty arises in distinguishing the one from the other. So long as the redness and vascularity are confined to the capillary vessels, or have their seat in the villous or follicular structure, there can be no doubt as to their inflammatory nature. It is only when they become more general and present the ramiform character, or when this character prevails, that any difficulty arises. This, however, is removed by an examination of the neighbouring veins, which, in mechanical congestion, will be found dilated, tortuous, or even varicose, according to the degree and duration of the obstacle by which it has been caused. The congestion of the veins may likewise be traced to its cause—to a tumour compressing them, to disease of the liver, heart, or lungs, which has obstructed the return of the blood through them. In inflammation the local congestion commences in the capillaries, afterwards extends to the small veins, but never to large branches; in mechanical congestion the blood accumulates first in the trunks, which are always conspicuous, and afterwards in the branches and capillaries. It is only when mechanical congestion is combined with inflammation, that the anatomical diagnosis becomes difficult or impossible. In strangulated hernia and intus-susception, we are certain of the existence of both kinds of congestion of the affected portion of intestine from the nature of the causes in operation, viz. compression or stricture on the one hand, and the influence of a morbid stimulus on the other. But when mechanical congestion, produced by a remote cause, is combined with inflammation, it is impossible to detect the existence of this latter disease, or at least to distinguish the one from the other with any degree of certainty. The same may be said of the combination of these two kinds of local congestion, under similar circumstances, in other organs.

With regard to local congestion from the influence of *depending position* during life or after death, it is only necessary to observe that no distinction can be drawn between it and that produced by inflammation when occurring in the same parts. But when local congestion exists in parts of organs which do not admit of the operation of this cause or of gravitation, and in the absence of a mechanical cause, we entertain

[*Inflammation*, 2.]

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no doubt of its being of an inflammatory nature, or the consequence of a morbid stimulus.

The redness observed in tissues in contact with the blood after death, produced by *imbibition*, and also occasioned by transudation, when decomposition has taken place, is easily distinguished from that which is the consequence of inflammation. It can never be confounded with that of inflammation when it occurs along the course of the larger subcutaneous veins; but it has frequently been so in the lining membrane of the large arteries especially, and of the veins. Whatever may be the circumstances which favour the red colour of imbibition, it never bears a near resemblance to that of inflammation. It is a mere dye, of a uniform, almost scarlet red colour, generally limited to the lining membrane, without any other perceptible change of the coats of the vessel; whereas redness from inflammation is of a dull, rather pink tint, extending more or less to the other coats, accompanied by a fine capillary injection of the subjacent cellular tissue, and marked congestion of the vasa vasorum; the lining membrane is softened and opaque, or easily removed; the cohesion of the other tunics is diminished; they are also thickened or swollen, and infiltrated with serosity or coagulable lymph.

I shall conclude this part of the subject by a few remarks on the *permanency* of the redness and vascularity of inflammation after death, as a character by which alone, and under doubtful circumstances, we may ascertain the existence of this disease, and distinguish it from the local congestions with which it may be confounded. And as the value of this character must, in part, be determined by ocular demonstration, it is only in cutaneous inflammations, and in those parts of the mucous membrane which are visible, that we can obtain positive evidence regarding it. The redness and vascularity of mechanical congestion, of position or gravitation, and of imbibition, differ, as we have seen, essentially from those of inflammation, in the mode of their production and other local circumstances. But they also differ essentially in other respects. Thus all these kinds of redness and vascularity are produced and maintained by appreciable causes which operate external to the vessels, without the blood or the vessels themselves undergoing any change by which the vital properties of either are so modified as to render the redness or vascularity permanent after death. By means of ablution, pressure or scraping, both of these physical characters disappear in a short time. In inflammation it is far otherwise, the employment of the same means never removing, or effecting only a slight diminution of either. Injections too, which penetrate the capillaries in the former kinds of congestion, cannot be made to reach them in that produced by inflammation. It is necessary, however, to observe, that this state of the capillaries, and the redness which accompanies it, and which constitutes what I mean by the *permanency* of these physical characters of inflammation, are more or less decided by the degree or stage of the disease. In the highest degree or second stage, the permanency of both is most marked; in the first stage it is much less, the redness diminishing or disappearing entirely after death, in slight inflammations of the skin of short duration. This difference in the permanency of the redness and vascularity of inflammation in its two stages may be explained by the facts already noticed, viz. the absence of coagulation of the blood in the first and its occurrence in the second stage, in which also the fibrine unites with and penetrates the capillaries, thereby retaining the colouring matter of the blood, and producing occlusion of the vessels. Although, however, I have said that *redness* disappears after death in slight inflammation of the

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skin, some degree of increased *vascularity* remains, as is seen by comparing the diseased with a healthy part. And besides, it is of great importance to know that even in such slight cases of inflammation, the cutis undergoes changes which render the existence and extent of the disease very conspicuous after the disparition of the redness, and from one to two days after death. The affected parts only of the skin assume a purplish tint, and become infiltrated with bloody serosity, and the epidermis is detached from these parts much sooner than from those which were not affected by the inflammation. This *post-mortem* congestion, and a more rapid tendency to decomposition than in ordinary circumstances, are conditions which ought not to escape the notice of the pathologist, when it occurs in internal organs. They are sometimes the only morbid appearances which are met with in fatal cases of scarlatina, and especially in rubeola, and indicate the extent not only of the inflammation, but of the depressing influence which it must have exercised on the vital function of these organs.

General Considerations on the Fluid Products of Inflamed Tissues.—The changes effected by inflammation in the quantity and qualities of the natural fluids of secretion, as well as the production of those fluids which originate in this morbid process, are subjects of great interest and importance when considered merely as signs by means of which, in the absence of, or in conjunction with, its physical characters, we are enabled to detect, during life or after death, its presence, or appreciate the degree or stage at which it has arrived. But as these changes may be considered as constituting in themselves distinct morbid states, it would not be consistent with the plan of this work to do more than enumerate the order in which they occur, and the stage or degree of the inflammation which they accompany. It has already been stated that the first change which is observed in the process of secretion is an increase in the quantity of the natural fluid of the affected part. In proportion as the inflammation increases in degree, the secretion diminishes, and is entirely suppressed in the second stage or most acute forms of the disease. During this progress, however, the qualities of the secreted fluids present important alterations. In inflammation of serous membranes, and at a very early period, the secreted fluid contains a quantity of albumen; afterwards, and as the inflammation increases, fibrine is added, and generally an admixture of the colouring matter of the blood, and lastly pus. The same order of succession is also observed to take place in the fluid products of inflamed mucous membranes. The mucous secretion, however, is, almost from the commencement of the inflammation, replaced by a serous fluid, which is often very abundant; this is succeeded by the presence of albumen and fibrine, and lastly of pus. The different degrees of fluidity, viscosity, and coagulability of the secretions generally of inflamed tissues are derived from the presence of the serum, albumen, and fibrine of the blood in various proportions. But the most important circumstances connected with these changes in the secretions of inflamed tissues, is the formation, or the separation from the blood, of two fluids of an opposite kind, viz. coagulable lymph and pus. The former, possessing vital properties, assumes, as is said, *spontaneously*, but no doubt in virtue of these properties, the solid form, becomes organized, and fulfils the all-bountiful purposes which nature has assigned to it in the economy of life,—the reparation of those injuries so frequently the consequences of the disease of which it is the product. The latter, possessed of no plastic properties, being as it were the residue of the former and of the molecular structure of organs, by a disorganizing or destructive process, is essentially inert, and like all substances

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incapable of being assimilated, is separated or removed from the body. The presence of these two products, therefore, marks not only the existence, but serves to distinguish the two most important periods of inflammation. Nor does this apply to the local conditions only of the disease; it is equally conspicuous in the general phenomena which accompany each period, or, as we have said before, each stage of inflammation, by the increased energy or vital power manifested in the first, and the diminution or perversion of the same in the second, and by the nature of the remedies required in each.

There is yet one circumstance to be noticed regarding the products of inflamed *mucous* membranes. Although sometimes assuming the form of membranous layers, cylinders, or tubes, they never become organized, never present the characters of the most simple of the Analogous Tissues, viz. cellular or serous tissue, so commonly met with in inflammation of serous membranes; and this is not owing to the influence of a physical cause, as their situation, but to a difference in their plastic properties. Under no circumstances have I ever met with a trace of bloodvessel in any of these forms of pseudo-membranous concretions. When the effusion which gives rise to them is accompanied by hæmorrhage, they are occasionally reddened or present streaks of blood. The most important of the forms which they assume are represented in Plate I. of the Fasciculus on Analogous Tissues.

DESCRIPTION OF THE PLATES.

PLATE I.

Inflammation of the cutaneous tissue. The figures represent the most important of the exanthemata, at that period of their progress in which the local characters, or elementary lesions peculiar to each, are most conspicuous. *Figs. 1, 2, 3*, represent the three principal forms of roseola. *Fig. 1*, Roseola *æstiva*, characterised by bright rose-red patches, single or in clusters, generally assuming a circular shape, the patches varying from the eighth to the fourth of an inch; the clusters from the fourth of an inch to one inch or more in diameter. *Fig. 2*, Roseola *autumnalis*, distinguished from the former by the defined circular shape of the spots, which vary from the fourth to three quarters of an inch in diameter, and by the dusky red colour which they present. *Fig. 3*, Roseola *annulata*, characterised by the rose-red colour appearing in the form of distinct rings, of a circular or oval shape, varying from two or three lines to an inch or an inch and a half in diameter, the inclosed skin appearing healthy. *Figs. 4, 5*, Rubeola: the first represents the ordinary form of the eruption, rubeola *vulgaris*; the second, the variety called rubeola *sine catarrho*. In *fig. 4* the efflorescence is seen to consist of small red circular spots of a papular character, and of larger patches, formed by the coalescence of these, of a semicircular or crescentic shape, the redness having a purple hue. Miliary vesicles sometimes appear in those parts of the skin where the inflammation is most severe. In *fig. 5*, which represents the disease when it occurs without the catarrhal symptoms, the crescentic form of the eruption is very conspicuous, the purple hue more marked, and less diffused than in the former. *Figs. 6, 7*, Scarlatina. *Fig. 6*, Scarlatina *simplex*, the bright red colour of the skin appearing in the form of broad, almost continuous, irregular, diffused patches, not unfrequently intermixed with miliary vesicles. The first stage of the eruption consists of innumerable red points, which increase rapidly in number, coalesce, and form the patches. *Fig. 7*, Scarlatina *maligna*, characterised sometimes by the deep lurid red colour of the efflorescence, and the occurrence of hæmorrhage or petechiæ of various extent. *Fig. 8*, the appearance of the tongue in

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scarlatina; the middle and posterior surface covered with a white pulpy exudation; the papillæ of the point and edges projecting, of a bright red colour, bearing a strong resemblance to a ripe strawberry. *Figs. 9, 10, 11, Erythema.* *Fig. 9* represents erythema *papulatum* and *tuberculatum*. A, erythema *papulatum*, the erythematous redness accompanied by small slightly elevated spots of the same colour, varying from the breadth of a pin's head to that of hemp-seed or a split pea; B, erythema *tuberculatum*, differing from the former only in the larger size and greater prominence of the circumscribed spots, grouped together in the diffused patches of the erythematous redness. *Fig. 10, Erythema nodosum*, distinguished from the former by the regular oval shape, larger size, and greater prominence of the patches. A, the appearance of the patches at the height, B, at the decline, of the inflammation. *Fig. 11, Erythema circinnatum* and *marginatum*; A, A, rings of the former, not elevated, and inclosing healthy skin; B, a patch of the latter, accompanied by papular redness, and presenting a slightly elevated and defined margin. *Fig. 12, Erysipelas* affecting the nose and eyelids, characterised by a deep fiery redness, intumescence of the skin, and phlyctenæ. *Figs. 13, 14, Urticaria.* *Fig. 13, Urticaria vulgaris* or *febrilis*, in the form of round wheels, A, A, and bands or stripes, B; the cutis pale, raised, and shining, and bounded by circumscribed redness; C, urticaria *conferta*, numerous wheels grouped on an inflamed basis. *Fig. 14, Urticaria tuberosa*, the wheels larger, confluent, and the swelling much greater than in the other varieties.

P L A T E I I .

Inflammation of mucous membranes. *Fig. 1*, a portion of small intestine showing the several forms of redness and vascularity of the inflamed digestive mucous membrane. A, A, ramiform injection, passing into the capilliform, B, B; the capilliform passing into the uniform, C, C; and the uniform passing into the maculiform or hæmorrhagic, D, D. *Fig. 2*, punctiform redness, A, A, of the villous structure of the ileum; giving rise to uniform redness, B, B, in patches. *Fig. 3*, mechanical congestion and inflammation of a portion of the ileum, in strangulated hernia. A, A, the mucous membrane of a uniform deep red colour; B, B, the projecting borders of the plicæ, and, C, C, of the mucous and submucous tissues, impregnated with pus; D, mechanical congestion of the external surface of the intestine; E, peritonitis. *Fig. 4*, chronic and acute inflammation of the colon. The chronic inflammation is indicated by the slate grey colour, A, A, of the mucous membrane; the acute attack which has supervened on the former, by the red colour, which, as in this case, is generally in the form of hæmorrhagic patches, B, B. *Fig. 5*, acute and chronic inflammation, and ulceration of the stomach. A, A, inflammatory redness occupying the plicæ; B, the star-like redness, and C, that in circles or rings, which accompanies inflammation of the follicles; D, chronic inflammation of the follicles, and, E, ulceration commencing in the same bodies. *Fig. 6, Bronchitis*; A, trachea; B, B, bronchi. The inflammation in this case was very severe, the mucous membrane being, nearly throughout its whole extent, of a high, uniform red colour, considerably thickened and softened.

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P L A T E III.

Inflammation of serous membranes. *Fig. 1*, Inflammation of the dura mater. The internal surface of the membrane represented in the delineation presents considerable redness and punctiform injection, the special seat of which is the fine cellular tissue connecting the arachnoid with the dura mater. At A, the redness is punctiform; at B, B, uniform, and giving rise in both to the formation of circumscribed patches of various sizes. The punctiform character indicates a severer degree or more advanced stage of the inflammation, and is the effect of the blood having penetrated the proper tissue of the arachnoid. This stage of the disease has been succeeded by suppuration, and the pus, C, is more abundant on this than on any other portion of the membrane. These appearances are extremely characteristic of the inflammation which succeeds to mechanical injuries of the head, for I have never met with them in any case of idiopathic inflammation of this membrane. *Fig. 2*, Inflammation of the pleura pulmonalis. A, A, a portion of the lung; B, the free surface of the pleura. The first and second stages of the inflammation are particularly well marked in this figure. The uniform redness, C, pervades a considerable portion of the membrane; and superadded to this is the punctiform injection, D, D, where the blood has penetrated and is retained in the substance of the pleura. *Fig. 3*, Inflammation of the peritoneum representing the several degrees of redness and vascularity, accompanied by the effusion of coagulable lymph. A, A, folds of the small intestine, along the contiguous borders of which the inflammation is most severe, as it always commences in these situations, and extends over the exposed surface of the intestine. The maculiform or hæmorrhagic stage of the disease is well seen at B, where the blood has penetrated the serous membrane, in the form of dark red points and patches. On the neighbouring fold of the intestine, C, C, C, are represented layers of coagulable lymph. In peritonitis, the inflammation generally commences, as has been stated, along the contiguous margins of the neighbouring folds of the intestine, and from thence proceeds over the surface in the direction in which the bloodvessels are distributed. It frequently does not extend backwards over the intestine towards the mesentery, the peritoneum included between the contiguous margins of the intestine, in this situation, presenting its natural colour, as at D, where the folds have been separated to shew this appearance. Hence it sometimes happens that the anterior half of the intestine presents all the physical characters of acute inflammation, whilst the posterior half is to all appearance healthy. *Fig. 4*, Pericarditis. A, the cut surface of the walls of the left ventricle; B, outer surface of the ventricle; C, pericardium. The morbid appearances represented in this figure are those of the hæmorrhagic form of inflammation of the pericardium, which generally occurs in consequence of an acute attack supervening to a chronic state of the disease. The membranous layers of coagulable lymph, which are generally very abundant, as well as the serous covering of the heart and pericardium, are penetrated with blood, the former being sometimes as red as if they had been left to soak in this fluid. The sanguineous effusion of the false membranes which have been detached and turned back, from the surface of the heart and pericardium, is represented at D, E, F, and G. *Fig. 5*, Pericarditis in an infant ten days after birth. The inflammation was very intense, as indicated by the deep, uniform red colour of the heart and pericardium. A, the heart; B, the pericardium laid open and turned aside; C, C, C, small patches

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of coagulable lymph. *Fig. 6 and 7*, Inflammation of the femoral artery from a dog after the application of a ligature. *Fig. 6*, external surface of the artery; A, superior portion much thickened and swollen by the accumulation of serosity and coagulable lymph, especially in the cellular sheath; B, a similar state of the inferior portion; C, D, inflammatory congestion of the vasa vasorum. *Fig 7*, internal surface of the artery; A, the pink redness of the lining membrane, terminating gradually in the natural pale colour. *Fig. 8* represents the uniform bright red colour of the internal surface of the carotid, from imbibition.

PLATE IV.

Inflammation of compound tissues or organs. *Fig. 1*, Pneumonia of the inferior lobe. The three stages of the disease, the congestive, plastic, and suppurative, or of active congestion, solidification, and suppuration, are remarkably manifest. The pale yellowish grey colour of the last stage is seen at A, where the substance of the lung is partly softened by the admixture of pus, and partly solidified by the coagulable lymph or fibrine with which it is impregnated; the red induration, hepatization, and granular character of the second stage are conspicuous at B; the deeper, more uniform red colour, and spongy aspect of the first stage are seen at C, passing into the healthy tissue, D, of the lung. *Fig. 2*, Hepatitis. Besides the diminution of consistence which accompanies inflammation of the liver, the blood is accumulated and retained in the affected parts, and obscures the lobular structure of the organ. The redness thus produced becomes deeper as the inflammation advances, until it is almost black. These appearances are seen at A, B, C, C, and D, D, at the surface and in the substance of the liver. *Fig. 3*, Nephritis. The tubular and cortical substances of the kidney affected with inflammation. A, the ureter laid open; B, B, cortical substance inflamed, the redness having a regular dotted appearance; C, C, the same substance infiltrated with pus; D, D, the inflamed tubular substance, terminating also in suppuration, E, E, E. *Fig. 4*, Inflammation of the pons Varolii and medulla oblongata. A, surface of the pons of a rose-red colour, presenting the punctiform and maculiform characters; the medulla oblongata, B, and the right motorius oculi, C, in the same state. *Fig. 5*, Inflammation of the cortical and medullary substances and membranes of the brain. A, A, capilliform and ramiform injection of the pia mater, terminating in the effusion of coagulable lymph, B; punctiform and hæmorrhagic characters of inflammation of the cortical substance, C, extending to the medullary substance, D, both of which are softened. *Fig. 6*, Inflammation of the mesenteric glands accompanying ulceration of the intestine in fever. A, portion of intestine laid open; B, B, ulceration of the glands of Peyer; C, mesentery; D, D, the glands enlarged, and in the first stage of inflammation; E, F, and G, other glands, presenting a progressive increase in the disease and its termination in hæmorrhage.

Plate I.
Fig 5.

Fig 1.



Fig 5.



Fig 4.



Fig 2.

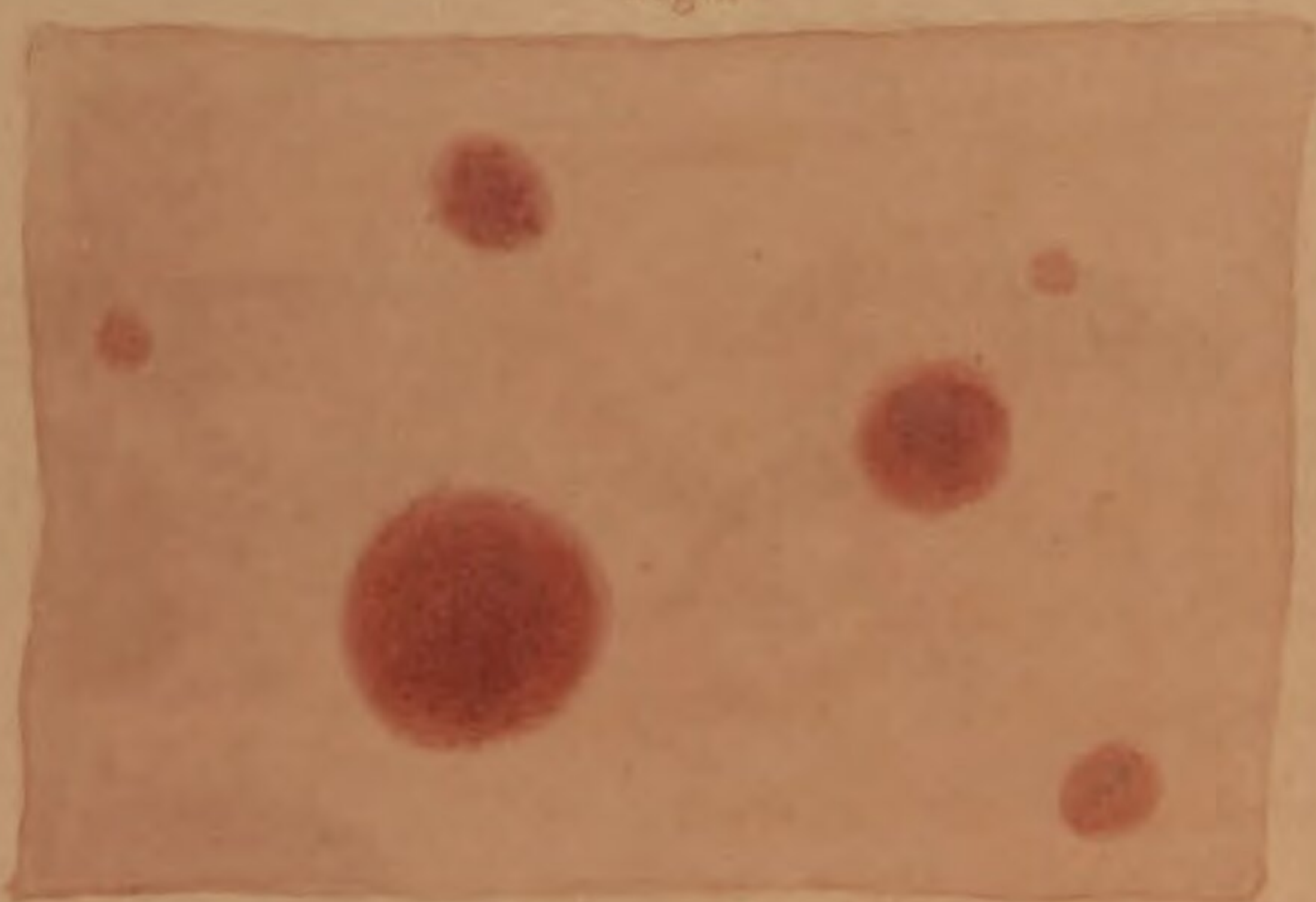


Fig 8.



Fig 6.



Fig 3.



Fig 7.

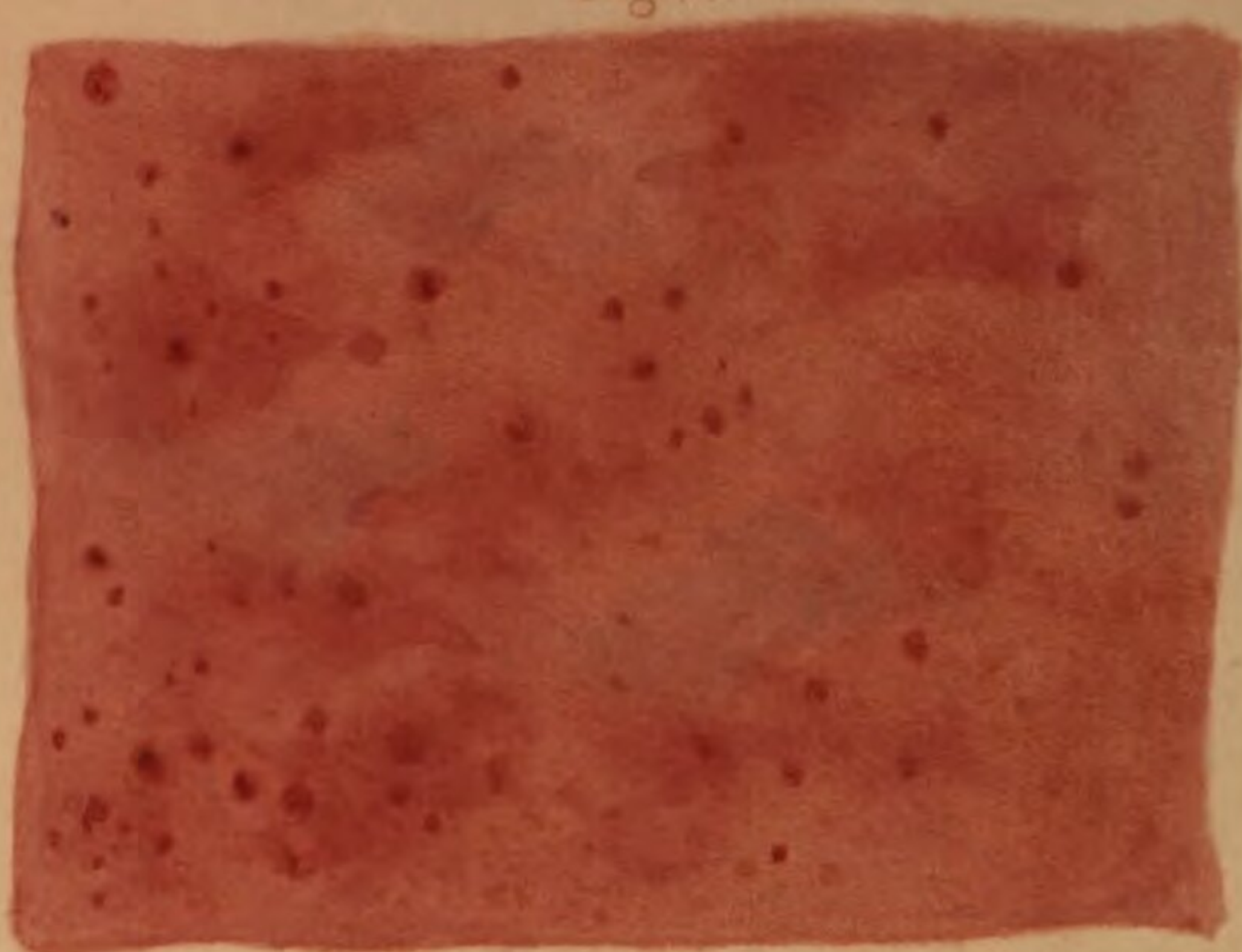


Fig 10.



Fig 11.



Fig 9.

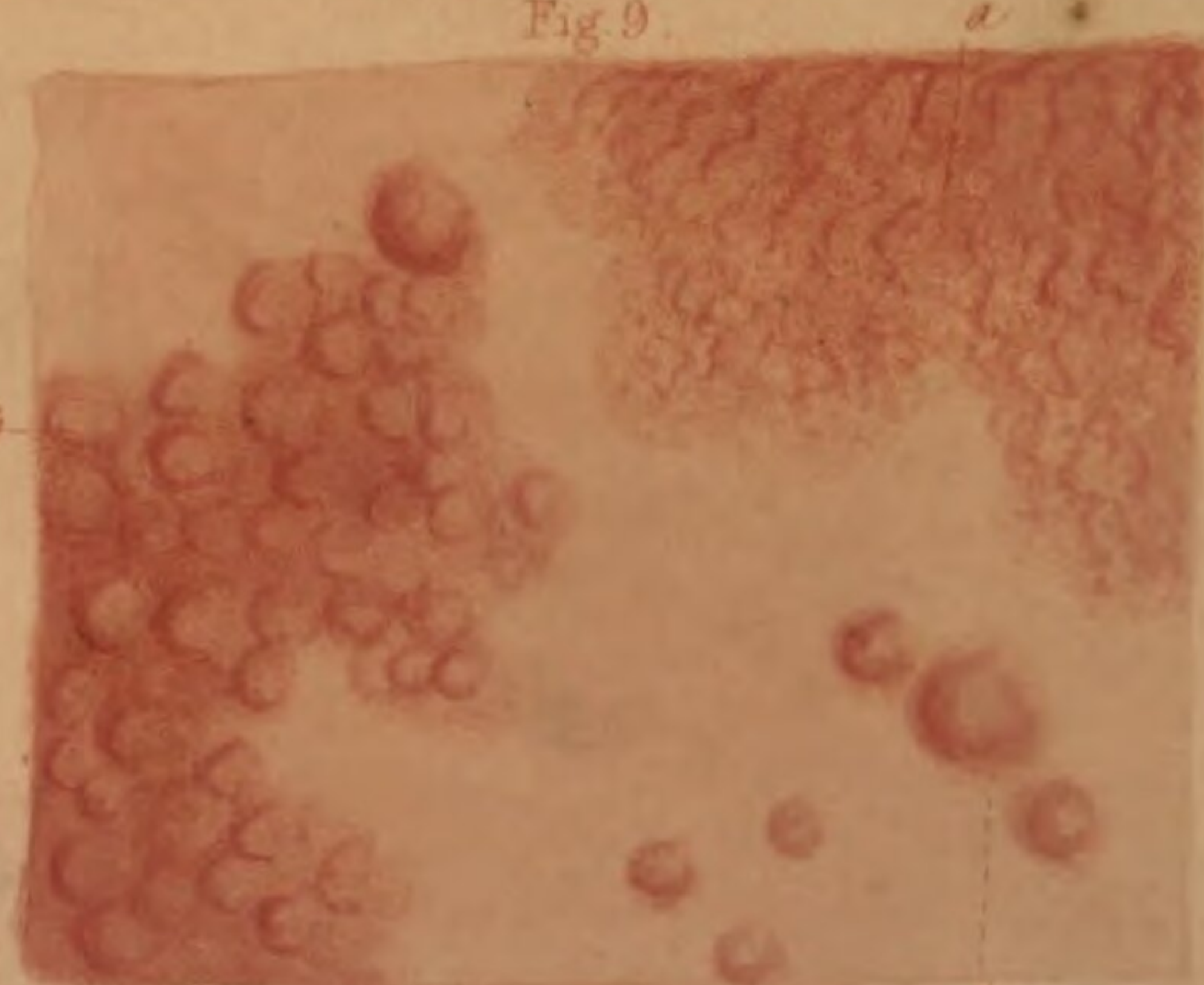


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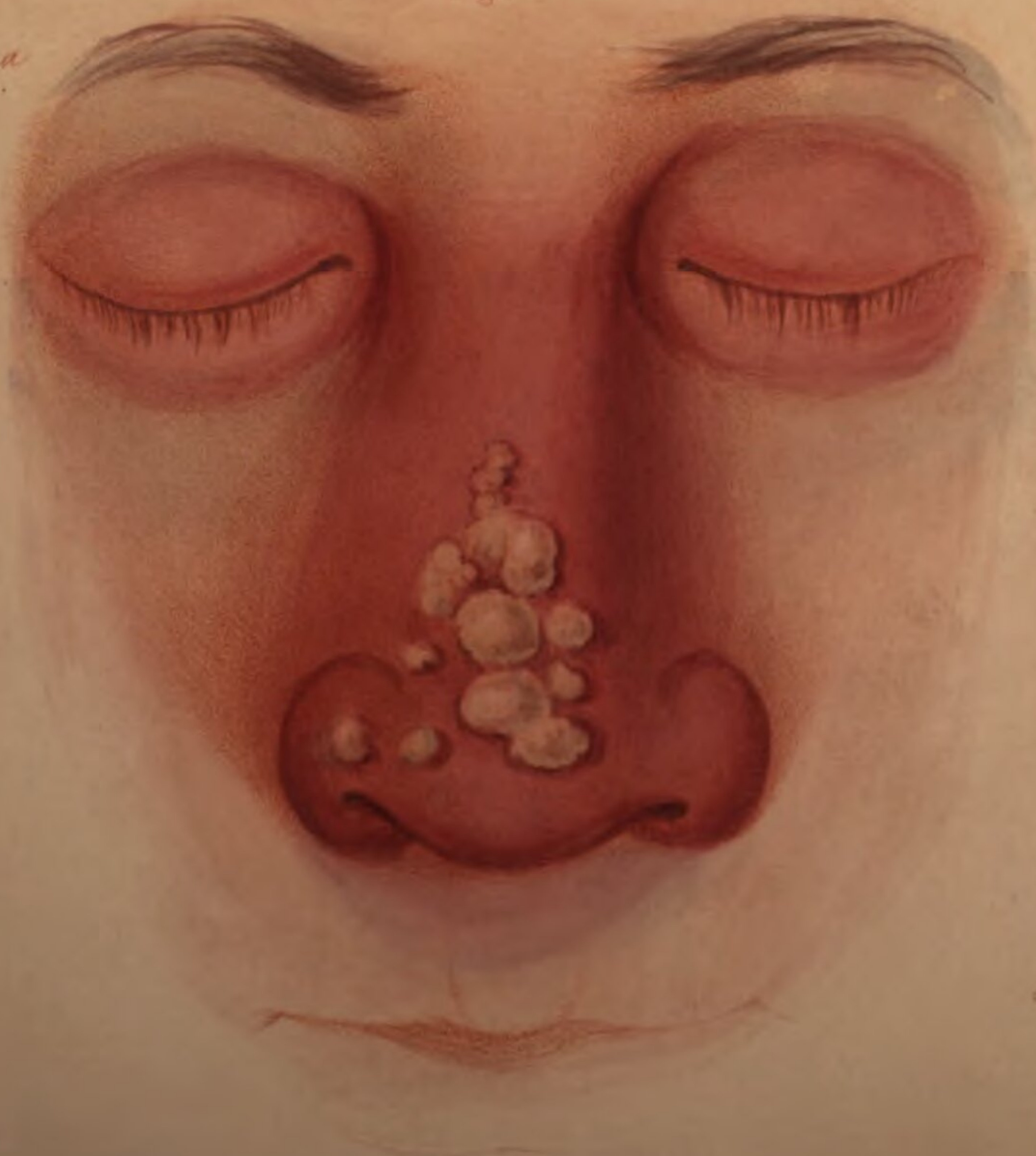


Fig 14.



Fig 13.

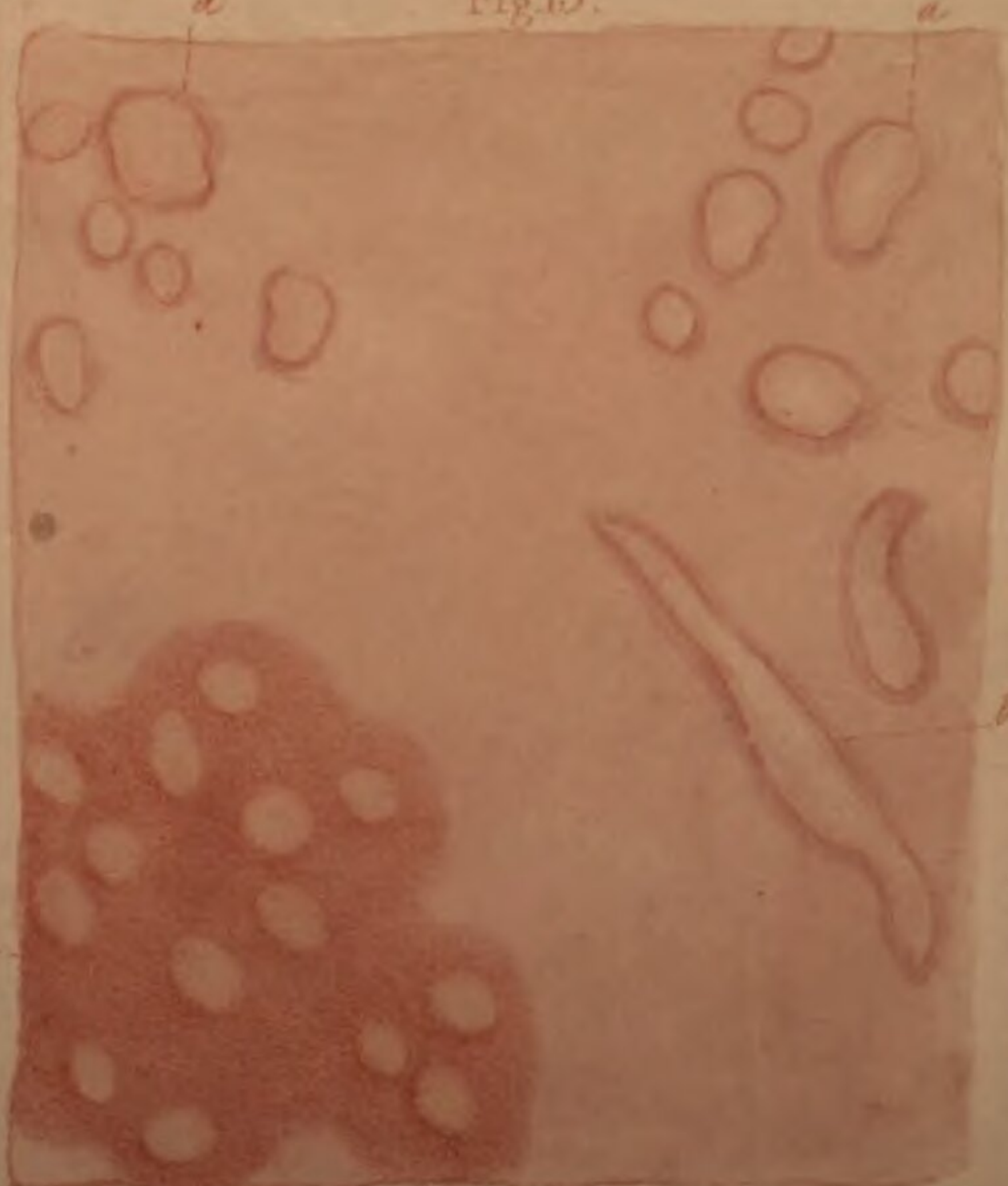


Plate II .



Fig. 1.

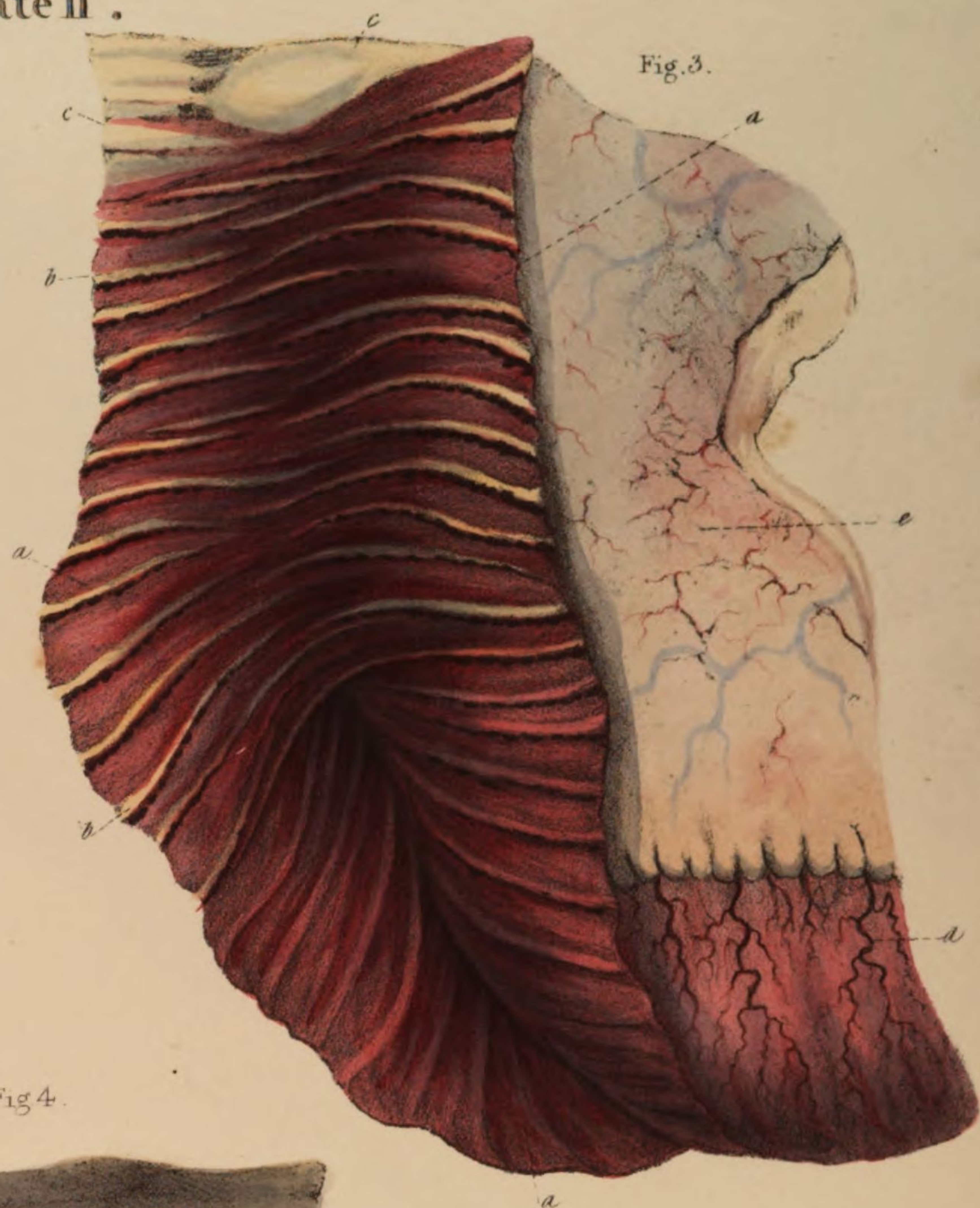


Fig 4.

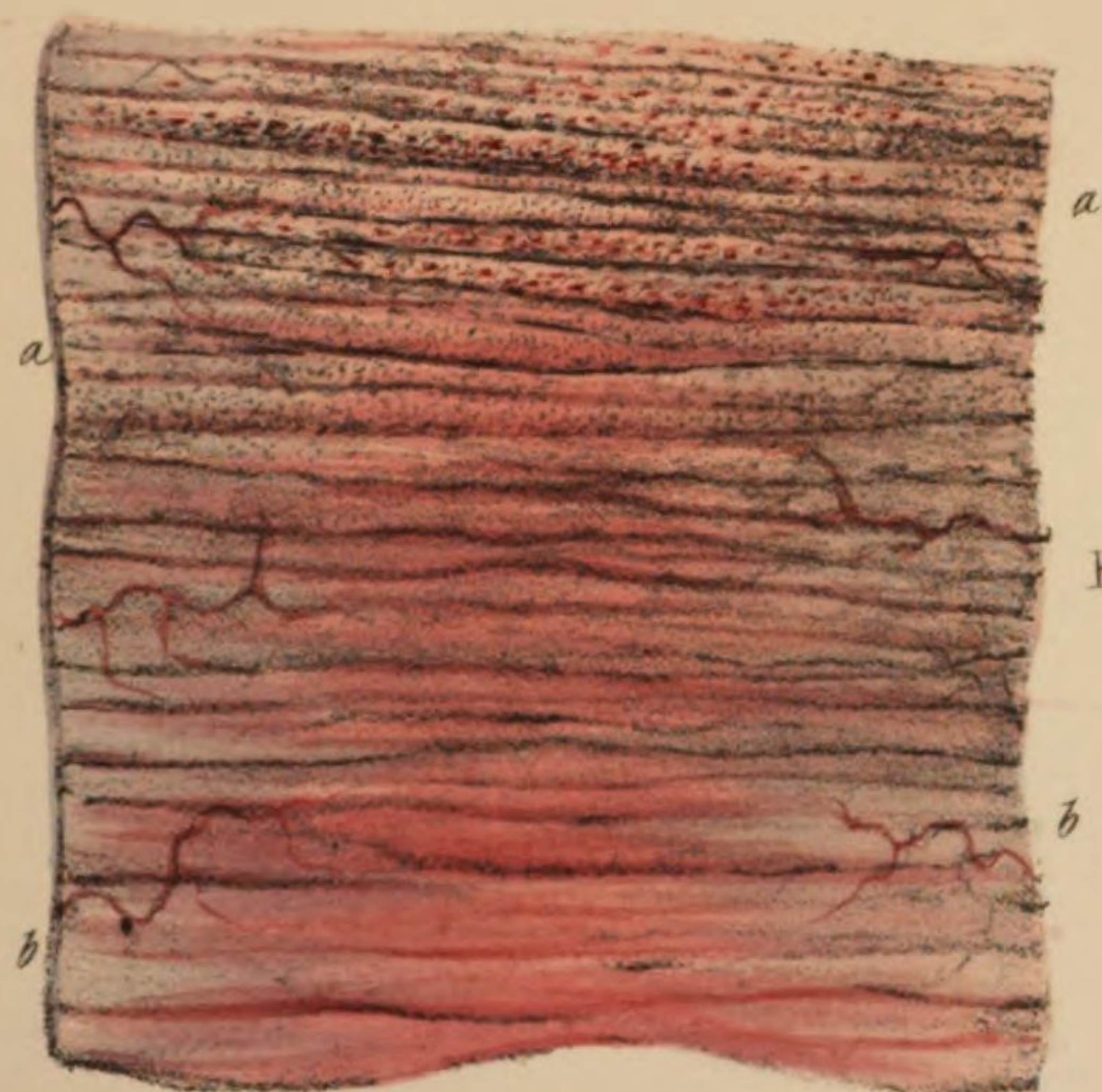


Fig 2

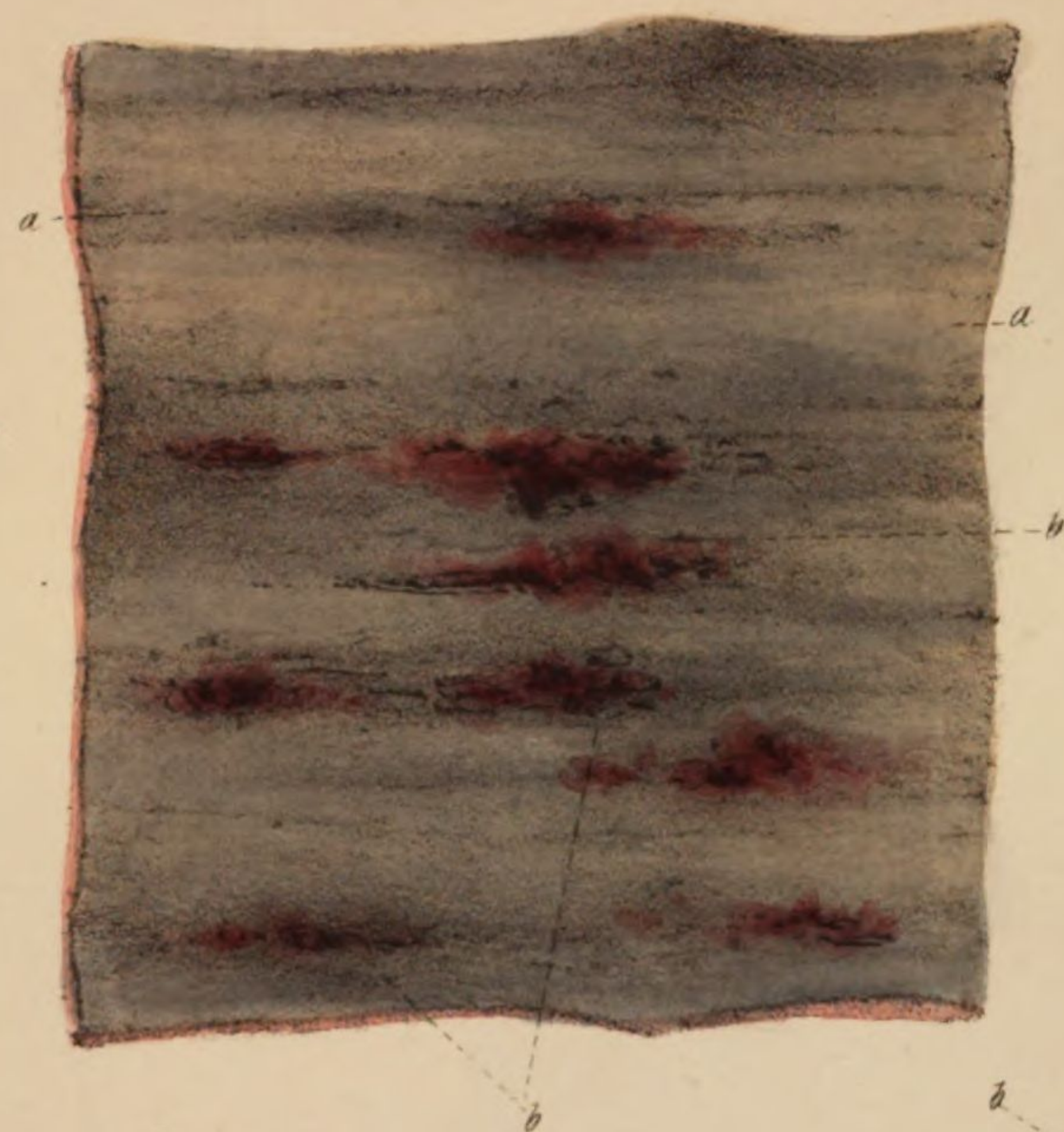


Fig. 5.



Fig. 6

Plate III.

Fig. 1.



Fig. 3.

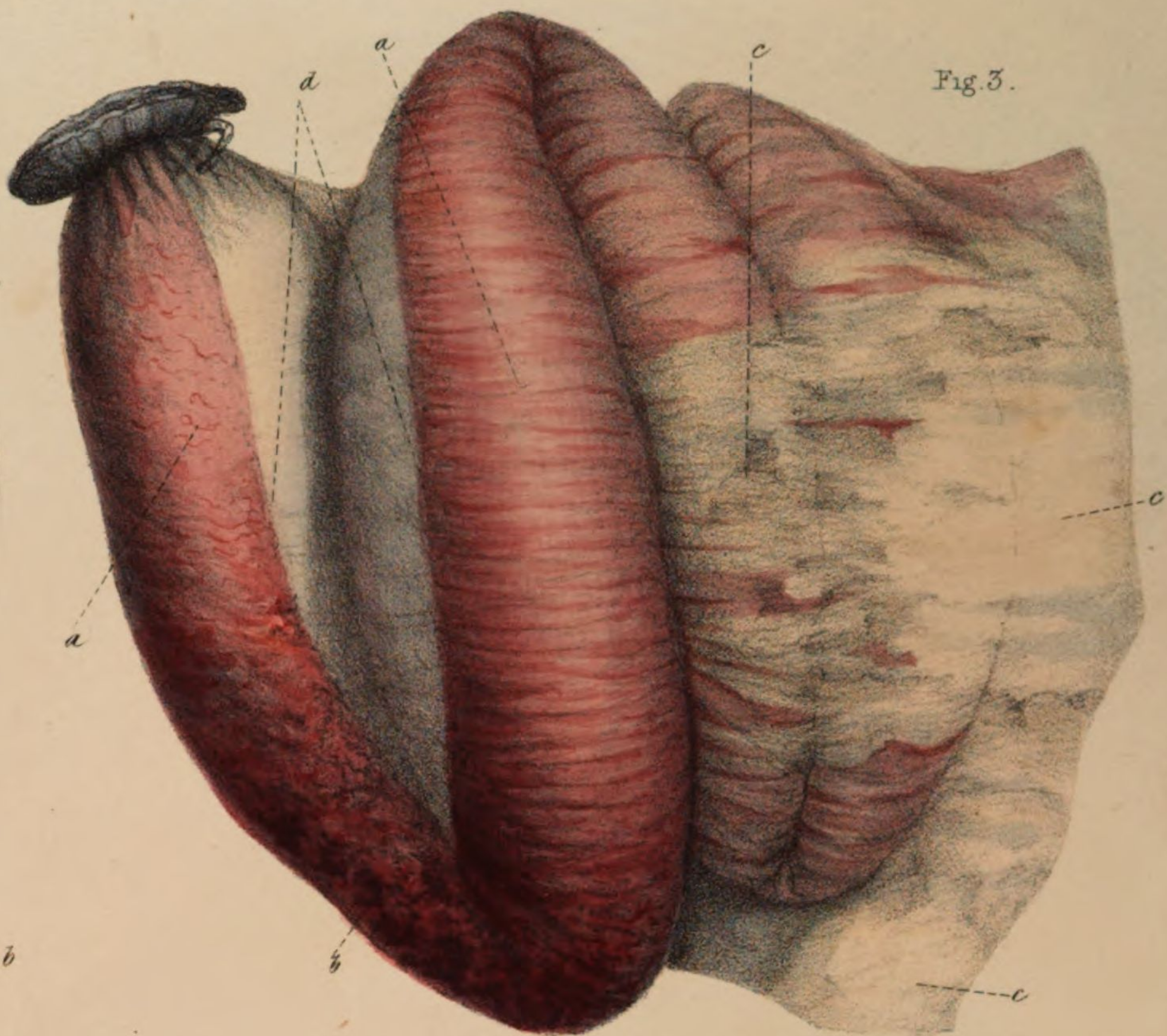


Fig. 4.

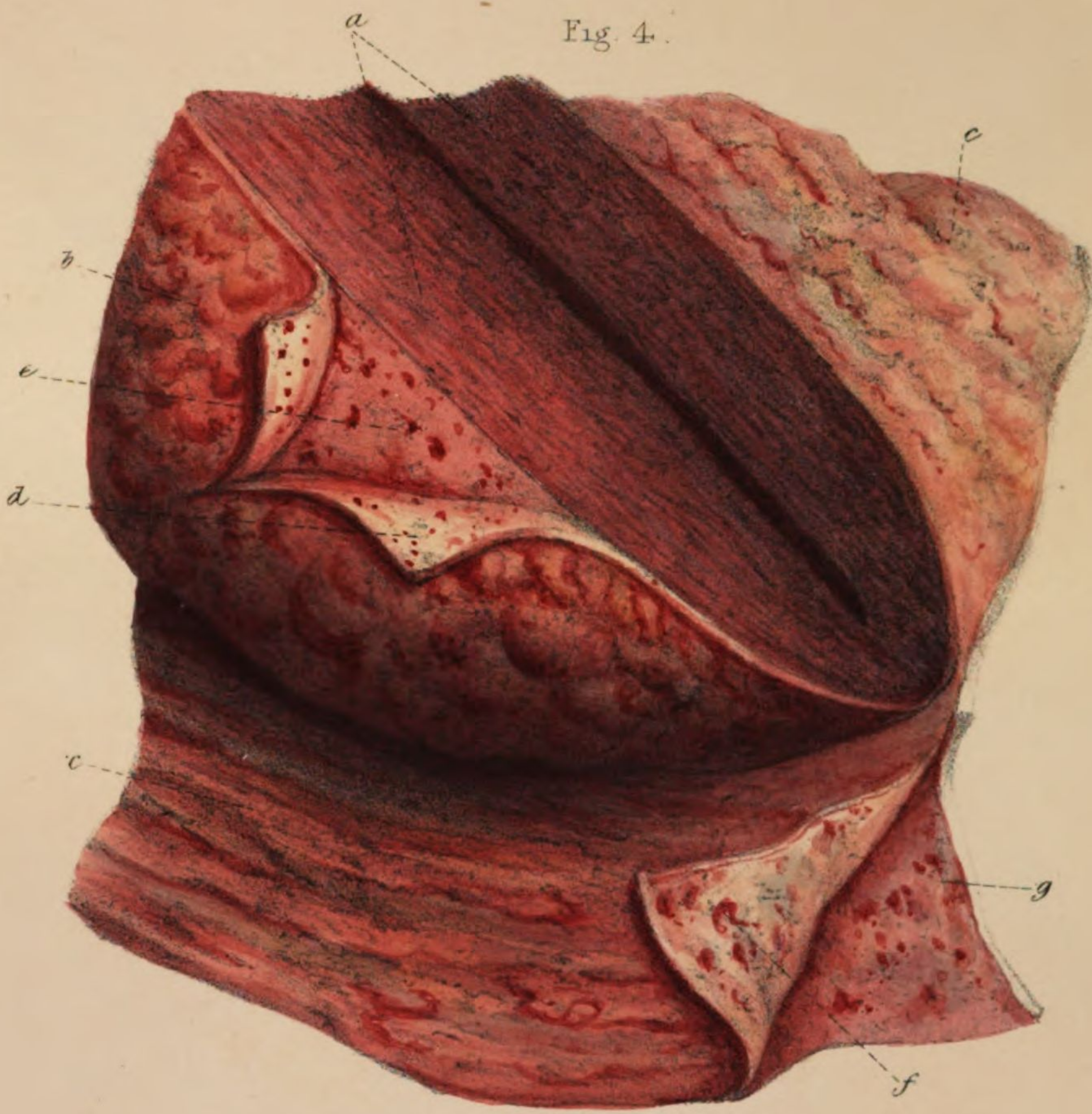


Fig. 5.



Fig. 2.



Fig. 8.

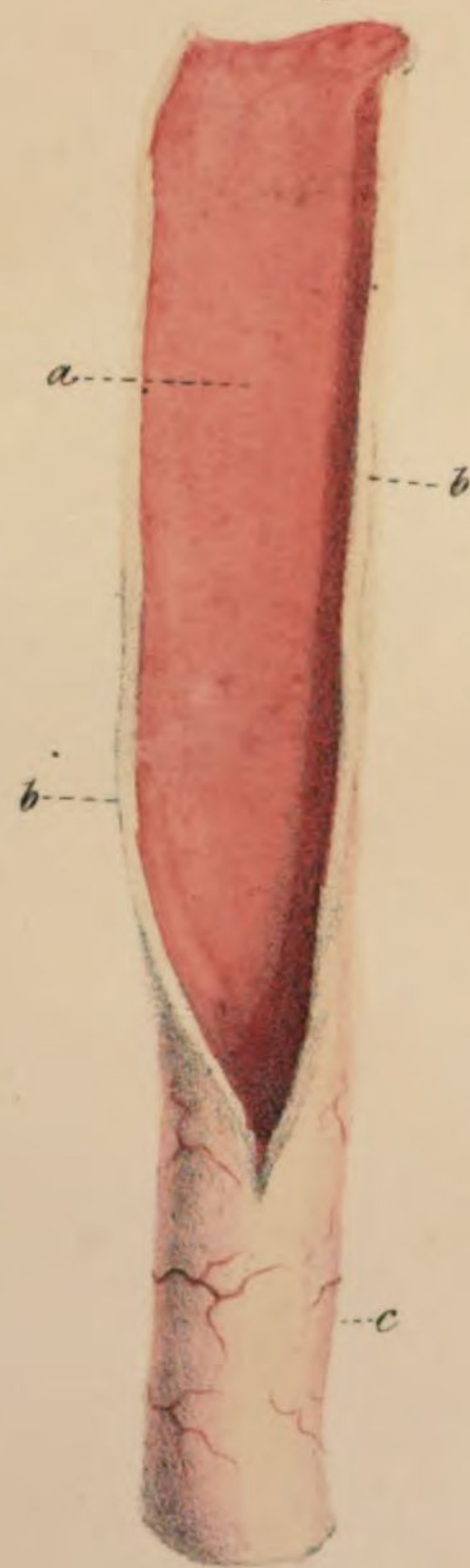


Fig. 7.

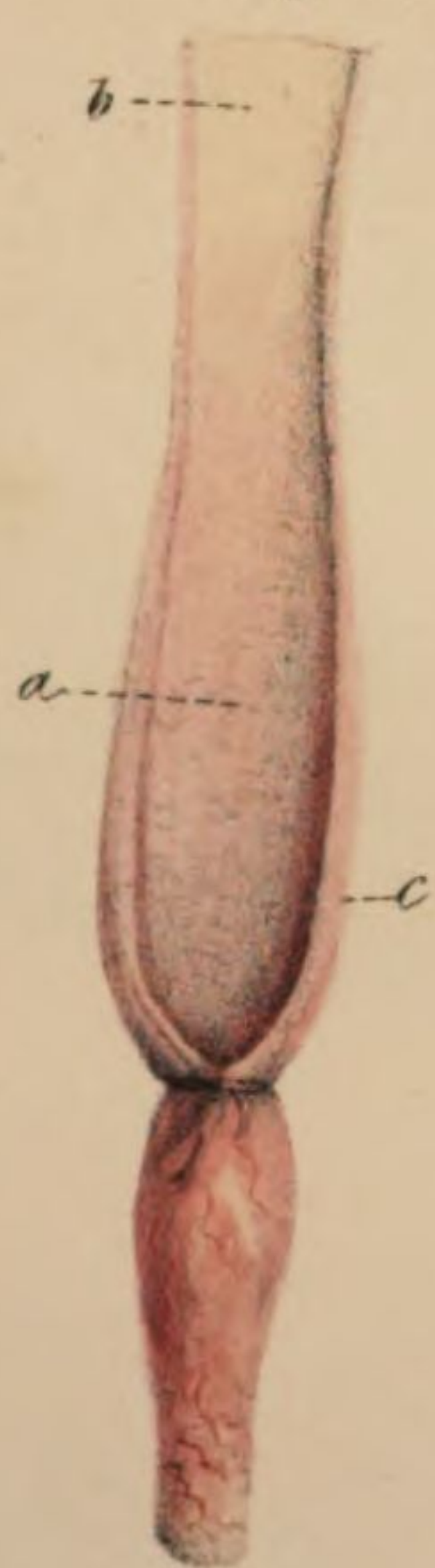


Fig. 6.



Plate IV.

Fig 1.



Fig 3.

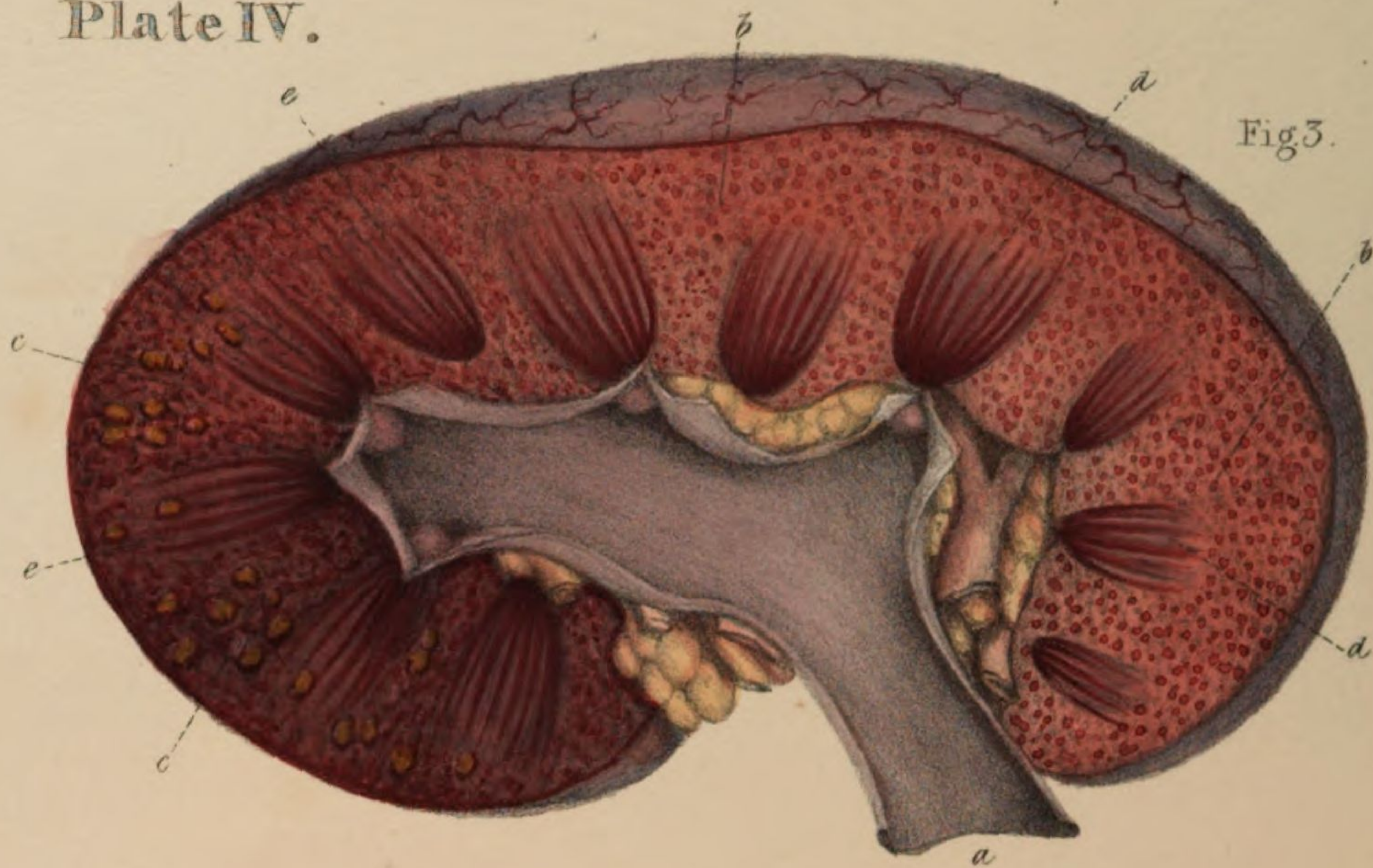


Fig 2.



Fig 4.



Fig 5.

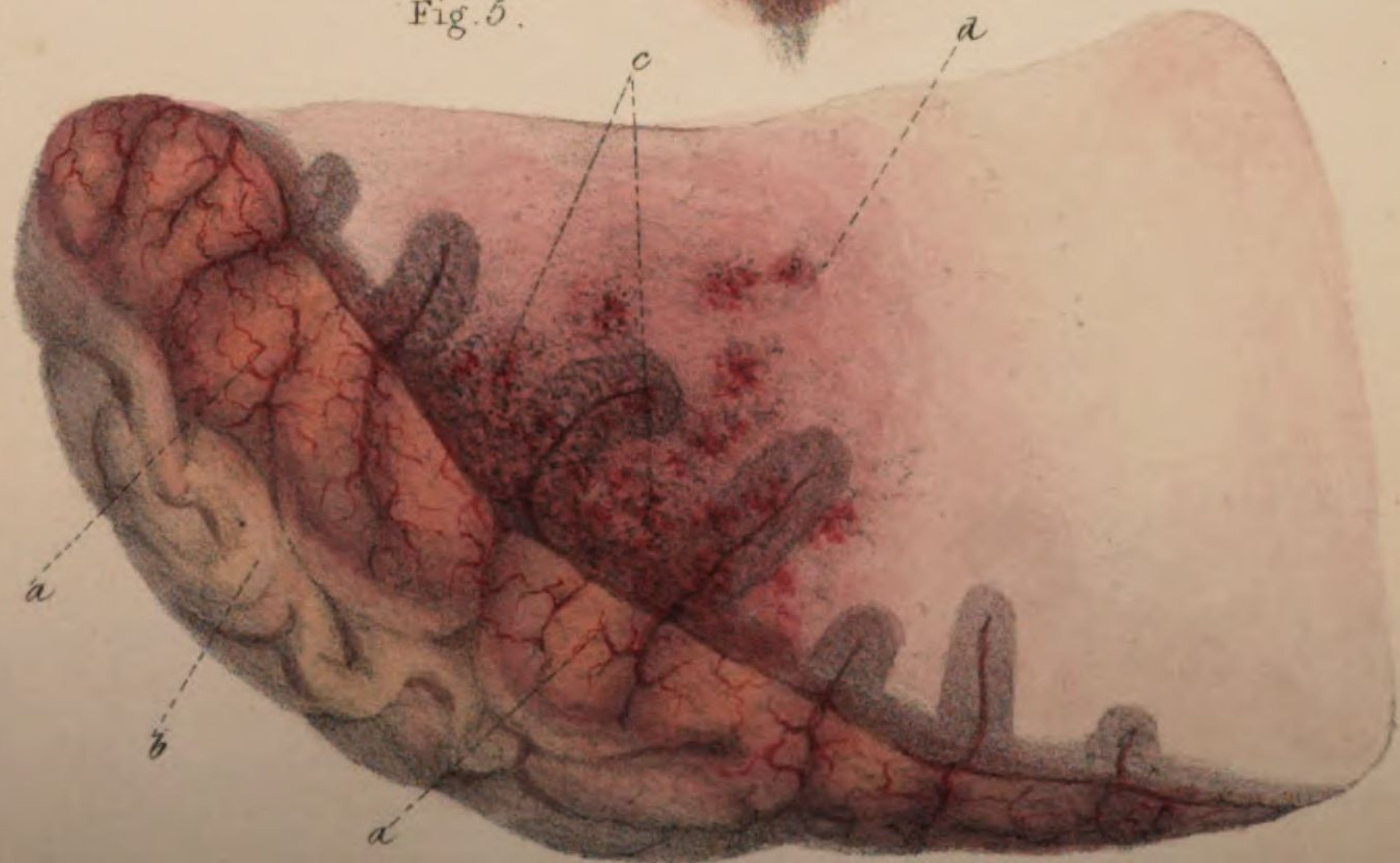
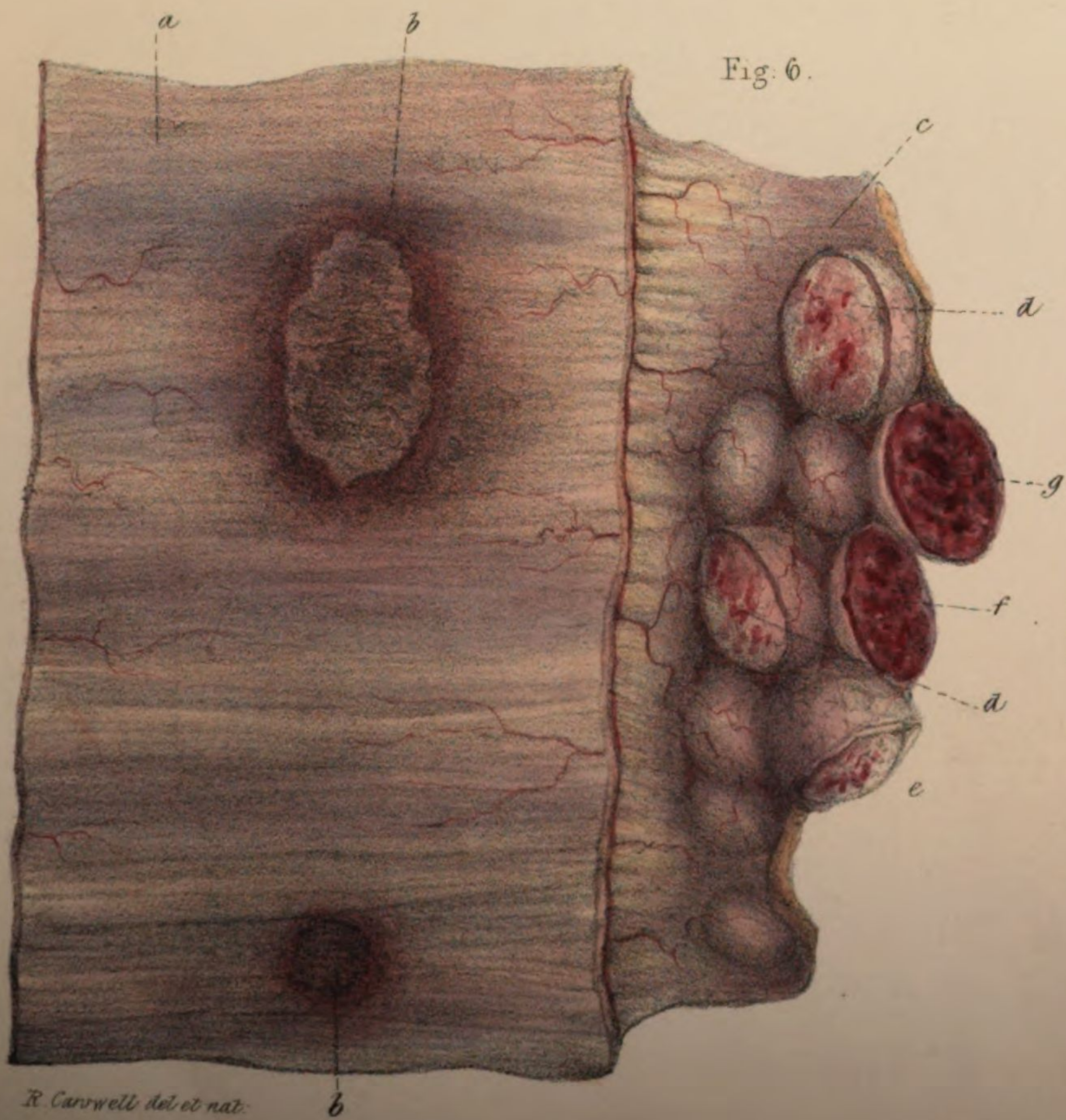


Fig 6.



ANALOGOUS TISSUES.

ALL solid, morbid products, which resemble the natural elementary tissues of the body, are called *Analogous Tissues*.

The number of these tissues nearly equals that of the natural tissues, as very few only of the latter have not been observed to form under those circumstances which give rise to the anormal development of all the others. The analogous tissues, including bloodvessel under the name of vascular tissue, are the following:—

- Vascular, including erectile.
- Cellular, including fat.
- Serous.
- Mucous.
- Cutaneous.
- Cuticular, including hair and nails.
- Fibrous.
- Fibro-cartilaginous.
- Cartilaginous.
- Osseous.

Before proceeding to describe each of these analogous tissues separately, I shall take a general view of the more important phenomena which relate to their origin and mode of development.

Origin of Analogous Tissues.—The analogous tissues present two important differences in regard to their origin. The plastic element of the blood, the spontaneously coagulable part of this fluid, or the fibrine, is by far the most frequent source, and furnishes the materials for the formation of the most perfect examples of the analogous tissues. It is to those which have this origin, that the term *analogous, accidental, adventitious, or pseudo-formations* is correctly applied. Another and entirely different origin of many of these tissues is a change taking place in the primary or existing elementary tissues, and even in organs, by means of which they are converted into tissues of a different kind, as for example, when cartilage is converted into bone, or cellular into serous or fibrous tissue. The analogous tissues which have this mode of origin are, in order to distinguish them from the former, called *analogous transformations*. Besides, the analogous formations, considered in reference to their mode of origin, require to be subdivided

[*Analogous Tissues*, 1.]

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into two distinct series. For although originating in the plastic element of the blood, they may be formed out of this substance, whether it be separated from the blood, and effused on the surface of organs in the state of coagulable lymph, in consequence of inflammation; or whether it be separated from this fluid which had ceased to circulate in its vessels, or had escaped from them in consequence of mere physical causes. Hence the important distinction to be established between the analogous formations derived from these two sources. In like manner the analogous transformations, although originating in the primary or general tissues, present this important difference, that they may be the result of the conversion of a tissue of a lower into one of a higher grade or more complicated structure, or the reverse, of a tissue of a higher into one of a lower grade or less complicated structure, thus constituting also two series of transformations, the first of which has been called the *ascending*, the second the *descending* series. The conversion of cellular into serous tissue, of fibrous into cartilaginous, or of the latter into osseous tissue, furnishes examples of transformation belonging to the first series; and the conversion of osseous tissue into cartilaginous, fibrous, and cellular tissues in succession, is an example of transformation belonging to the second series. On these principles I shall establish two great divisions of the analogous tissues, viz.—Analogous Formations and Analogous Transformations. Of the former, I shall treat, first, of those which originate in the *fibrine* of the blood, in the absence of inflammation, or at least in the absence of what is called *coagulable lymph*, as a product of inflammation; and afterwards of those which originate in the same substance when a product of this pathological state.

ANALOGOUS FORMATIONS *originating in the fibrine of the blood*.—The evidence in support of the doctrine which maintains that analogous formations may originate in the fibrine of the blood, is derived from the changes which are observed to take place in the blood which has ceased to circulate in the heart or bloodvessels, or which has been effused into the substance of an organ. The cessation of the circulation which precedes these changes in the blood, may be effected by the operation of mechanical, physical, and vital agents, which act either on the blood itself, or on the vessels in which it is contained. The most obvious examples of the operation of the first kind of agents are those in which the circulation is at once arrested, either in an artery or a vein, or in both, by the application of a ligature; or those in which the blood stagnates in the veins, for example, of the inferior extremities, from a mechanical obstacle to its return, situated in a remote part, as in the heart. The first change which the blood is observed to undergo in these circumstances is coagulation, the extent of which, in an artery, is almost always determined by the situation of the first branch of considerable size, sent off from the obstructed vessel, between the ligature and the heart, but which, in the veins, varies with the situation of the obstacle, and the greater or less facilities afforded for the development of a collateral circulation. Whatever may be the extent of the coagulation, the subsequent changes which take place in the blood are as follow:—The coagulum acquires gradually an increase of density, which is accompanied by the removal of the red colouring matter of the blood. The fibrine becomes thus more and more apparent, and is recognised by its pale straw-colour, and more especially by the manifestation of its plastic properties, whereby it assumes, almost from the commencement, a laminated or fibriform arrangement. In this, the early stage of what may be called the process of organization of the fibrine, there is one circumstance which is peculiarly interesting, not only because it enables us to explain the origin and mode of formation of some analogous

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tissues, but because it shews that the vital endowments or plastic properties of fibrine, under the circumstances in which we are now considering it, are of the same kind as those of coagulable lymph, however much they may vary in degree. The circumstance to which I allude is the tendency, from the commencement, of the fibrine to escape towards, and to accumulate at, the circumference of the coagulum, or to place itself in contact with the living tissues which surround the coagulum. It is not only in a blood-vessel that this is observed; it is seen to take place in the cavity of the heart in the formation of what are called polypi, whatever may be their mode of attachment, as well when they occupy the greater part of one of the cavities of this organ, as when they are small and connected only by a narrow pedicle; and sometimes in large serous cavities in the case of hæmorrhage, when the patient has survived that accident for a certain length of time. One of the most remarkable cases of this kind which I have met with, occurred in a young officer who died about three weeks after having received a wound of the cœliac artery. Besides a considerable quantity of blood contained in the retroperitoneal cellular tissue, there was also about a pint in the cavity of the pelvis. In this situation it was partly fluid and partly coagulated, but the whole was enclosed in a shut sac, varying from one to two lines in thickness, composed of several layers, and of sufficient consistence to permit of its being separated and removed entire from the parts with which it was connected. That this peripheral, or eccentric, disposition, as it may be called, of the fibrine, succeeding to coagulation of the blood, is a phenomenon which constitutes a part of the process of organization of this plastic substance, will become more obvious as we proceed.

This process of organization of the fibrine may go on throughout the whole of the coagulum, or it may stop at some distance from the central portion of it. In the first case, the vessel becomes permanently obliterated, for the fibrine, as it acquires a more perfect organization, unites with the internal surface of the vessel, contracts gradually, and converts it into a fibrous cord. In the second case, the vessel may remain pervious to a limited extent, for the unorganized central portion of the coagulum, or the serum and red colouring matter, or, as sometimes happens, a grumous milky-looking fluid, found in this situation, may be slowly impelled onwards by the same contractile property of the eccentric tissue which produced the cord-like obliteration of the vessel in the former case, and thus the admission and circulation of the blood be re-established. These remarkable and important changes are strikingly illustrated by the appearances represented in Plate II, *figs.* 5, 6, and 7. The vessels, or rather canals, observed by Blandin and Lobstein in the fibrinous coagula of arteries which had been obliterated by ligature some years before, were probably of this nature.

Besides the eccentric disposition, the laminated and fibriform arrangement of the fibrine, and its ultimate conversion into a cellulo-fibrous tissue of various degrees of density, forming an intimate union with the parietes of the vessel in which it is contained, there is yet to be noticed the vascular organization of this product, which is frequently not less conspicuous than the other manifestations of its vital properties. The vascular organization of the fibrine takes place under two conditions:—first, when the circulation has been arrested by a mechanical cause which has no tendency to induce inflammation in the obstructed vessel, and the only appreciable organizable product is the fibrine of the coagulum; secondly, when the circulation has been arrested by a ligature, or by inflammation of the vessel, and the organizable product occurs not only in the form of fibrine, but also in that of coagulable or plastic lymph.

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Although in both these conditions I have several times satisfied myself of the vascular organization of the fibrine, I have never been able to detect the primary and independent formation of bloodvessels in this substance. On the contrary, I have always found the vessels which it contained existing in the form of capillaries, derived from the collateral branches of the obstructed or obliterated vessel; for they were not only continuous with the latter, but, penetrating the fibrine at the circumference of the vessel at various points by a single branch, afterwards assumed a capillary distribution in the laminated and fibriform structure of this substance. In the first of these conditions the capillaries found in the organized fibrine are, perhaps, always few in number, whereas in the second they are sometimes very numerous, and so conspicuous that no kind of injection is required to demonstrate them. That the formation of these vessels in the latter conditions is favoured to a great extent by the presence of the plastic lymph which is thrown out by the inflamed vessel into its cavity, and some of which appears even to penetrate the recent fibrine, there can be no doubt; but that their formation in the former is entirely dependent on the presence of the same product is an opinion which, although supported by the broadest analogy, is opposed by a multitude of negative facts. It is by no means rare to meet with the fibrine of the blood, particularly in veins, sometimes of large size, presenting the several grades of organization which I have described, adhering with various degrees of tenacity to the internal surface of the vessel, and presenting sometimes a few, at others no trace of capillary vessels. There is no plastic lymph to be seen within the vessel, and no modification of its walls, except an increased development of the vasa vasorum, and sometimes diminished cohesion of the coats the one to the other. If these two changes are to be regarded as evidence of inflammation being present in a slight degree, still there is no plastic lymph to be perceived, although the fibrine has assumed the cellular and cellulo-fibrous structure. This, therefore, if these circumstances have been correctly observed, must have been accomplished by the inherent properties of the fibrine itself. It is indeed highly probable that the presence of this substance, acting to a certain degree as a foreign body, induces a modification of the capillary circulation, and, consequently, of the nutrition of the walls of the vessel with which it is in contact, such as the softening of these and the injection of the vasa vasorum alluded to, without which we could not explain the union and vascular connexion which take place between the containing and contained parts.

From these facts, and the reasoning founded upon them, we should be led to conclude that the fibrine of the blood, like the plastic lymph of inflamed surfaces, is, under the circumstances specified, susceptible of organization, of becoming united with contiguous tissues, and of undergoing those changes, in the progress of its development, which usually characterize the analogous formations; that it undergoes these changes either along with the plastic lymph which is effused in inflammation, or alone when no plastic lymph appears to follow the presence of this pathological state.

The vascular organization of the fibrine of blood which has been effused into the substance of an organ is best exemplified in cerebral apoplexy, when, after several attacks, an opportunity is afforded us of observing, after death, the successive steps of this process; but as the reader will find this subject illustrated in the Fasciculus on Hæmorrhage, I shall proceed to notice, generally, the formation of what are called polypi of the heart and arteries, and of phlebolites, as they furnish additional examples of the process of organization, although in a less degree, arising in the fibrine of the

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blood. I shall not stop to refute the reasoning of those pathologists who attribute the formation of polypi of the heart exclusively to inflammation of its lining membrane, or, as it has been aptly called by M. Bouillaud, *endocarditis*. The size, form, and mode of attachment of these polypi are inconsistent with such a mode of origin. They vary from the size of a cherry to that of a walnut, are generally of a globular or ovoid form, and are always fixed by numerous prolongations interwoven with the muscoli pectinati, or smaller columnæ carneæ. They are usually of a deep red colour externally, probably from imbibition, are composed of fibrine of various degrees of consistence, at first of a reddish brown, afterwards of a reddish or greyish yellow colour, and contain sometimes one cavity of considerable size, at others several smaller cavities, filled with a fluid resembling turbid serum, a milky-looking or grumous fluid. I have sometimes found them composed of fibrine and cellulo-fibrous tissue, the latter being most apparent at the circumference, and occasionally assuming a laminated, but more generally an irregular fibriform arrangement. They have never presented any trace of bloodvessel to me, although some pathologists state that they have succeeded in injecting them. See Plate II. *figs.* 1 and 2.

With regard to the origin of these polypi, I shall only observe that it has been generally traced to an inflamed state of the blood in general, and stagnation or retarded motion of this fluid in the cavities of the heart. Almost all the cases I have met with of polypi in this organ, and the most of those recorded by authors, have occurred under these conditions of the blood. The far greater frequency of their formation in the auricles than in the ventricles, and in that portion of the auricles furnished with the muscoli pectinati, are circumstances which accord perfectly with this view of their origin. I do not, however, include under these remarks the formation of those morbid products called excrescences and concretions, which, although favoured by the conditions of the blood alluded to, depend essentially on the presence of inflammation of the lining membrane, of the valves in particular, of the heart, and to which they are attached in such a manner that the fibrine could not have been retained by any physical condition of these parts.

The conversion of the fibrine of the blood into fibrous, cartilaginous, and osseous formations, is also sometimes observed to take place in aneurisms of long standing, in those layers of this substance which occupy the circumference of the sac, and which then bear a striking resemblance to the diseased coats of the artery. The formation of what are denominated *phlebolites*, is a remarkable example of the gradual conversion of the fibrine of the blood in the veins, into isolated round or ovoid bodies of a stony hardness. These bodies vary from the size of hemp-seed to that of a pea; they consist at first of a clot of blood, which, as it loses its red colour, assumes all the physical characters of fibrine. This is disposed in several concentric layers, which acquire considerable density, approaching occasionally to that of fibrous tissue, without, however, presenting its other physical characters, and always terminating in a degree of hardness equal to that of stone. In this state, as well as at their commencement when in the form of a fibrinous coagulum, they are contained in a serous envelope, and lie merely in juxta-position with the lining membrane of the vein. Plate III. *figs.* 1 and 2, represent them in the several stages of their formation in the broad ligament of the uterus, where they are generally found.

I shall only further illustrate this interesting part of our subject by a passing notice of those formations which arise in the fibrine of the blood in its course through the

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arteries, apparently under the influence of a physical agent; but of what kind, or the manner in which it operates, is not known. Thus, needles which have been made to transfix an artery, become as it were encrusted with fibrine; spiculæ of bone projecting into the cavity of an artery, have sometimes appended to them pediculated pyriform masses of fibrine, an inch in length, half an inch in breadth, and an eighth of an inch in thickness. They present a fine radiated structure, and lie in the direction of the vessel with their basis from the heart, having received this direction from the current of the blood during their formation. An extremely interesting example of this kind is represented in Fasciculus VII. Plate III. *fig. 2*. It is on the same principle that a coagulum forms around a mesh of fine thread when introduced into the cavity of an artery, as in the case represented in Plate III. *fig. 3*. The experiment was performed on a dog, in the following manner, by M. Magendie:—After laying bare a portion of the axillary artery, a needle, armed with several fine threads, was introduced obliquely through the walls into the cavity of the vessel. The point being carried upwards, it was withdrawn in that direction, and the thread pulled till its extremity dropped within the vessel, when it was left floating in the stream of blood. On the following day the circulation through that part of the vessel had ceased, and on the third day the animal was killed. Externally, that portion of the vessel submitted to the experiment was red, swollen, and felt firm, and, when laid open, contained a fusiform coagulum, about an inch and a half in length, suspended near its middle by the thread, enclosed within it, and passing through its centre in the direction of the circulation. The lining membrane of the artery was not inflamed, nor was there the slightest trace of plastic lymph.

ANALOGOUS FORMATIONS *originating in the plastic lymph of the blood*.—Having disposed of the origin and mode of development of the analogous formations derived from the fibrine of the blood, we have now to consider the same important subjects in regard to those which arise in the coagulable or plastic lymph of this fluid. As the plastic lymph which is separated from the blood and effused on the free surface of organs, affords the best means of studying the changes which it undergoes, and as these changes are most conspicuous on the free surface of serous membranes, I shall describe them as they occur on the pleura or peritoneum.

One of the most remarkable properties of plastic lymph is that by which it coagulates spontaneously when effused, a property which is believed to reside in the fibrine of which it is found by chemical analysis to be essentially composed. A still more remarkable property of the plastic lymph, which, as well as the former, is of a vital nature, is that by which it becomes subservient to the formation of more perfect products, by which it forms bloodvessels for the growth and development of these, and to connect them with the living tissues of which they afterwards become a part, in order that along with them it may contribute to the reparation of the injuries induced by accident or disease. Whether the organization of plastic lymph be accomplished on the surface of a serous membrane, or on that of a solution of continuity; whether in the form of membrane or granulation; when a loss of substance is repaired by the reproduction of a cutaneous, mucous, bony, or other tissue, the process is essentially the same in all. As the formation of bloodvessel is the first and most important of the vital phenomena which present themselves in the process of organization of the plastic lymph, the consideration of it merits the first place in a description of the analogous tissues.

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Formation of Bloodvessels.—In describing the formation of bloodvessels, we have to consider the changes which take place not only in the plastic lymph, but also in the inflamed serous membrane of which it is a product, and between which and the former, a vascular connection is established even at an early period of the development of the analogous formations. Besides the redness and vascularity which characterize inflammation of the pleura, and which appear at first in isolated spots situated in the subserous cellular tissue, but which, at a subsequent stage of the inflammation, extend to the serous membrane itself, the free surface of this membrane presents a villous or granular appearance, particularly at those points where the redness and vascularity are most marked. The pleura has now undergone important changes, especially at these points; it appears considerably increased in thickness, is so soft as to be easily removed by passing the edge of a scalpel over it, and contains small specks or striæ of blood. This state of softness, which follows as a necessary consequence of the modification of nutrition induced by inflammation of every tissue, increases with the progress of the inflammation, and in all probability not only precedes its extension to the pleura, but also places this membrane in a condition to receive the plastic lymph which is effused, and to afford a ready passage for the enlarged capillaries through which the blood is to be conveyed to the vessels of the new membrane which is to become as it were engrafted upon it. That this process of softening of the pleura is absolutely necessary to the vascular union which takes place between the general circulation and that of the false membrane, will appear more obvious when we reflect that the extremities of a fractured bone take no part in the reparation of the injury, until they have become softened by inflammation and penetrated by plastic lymph.

The plastic lymph which forms on the surface of the pleura appears first in the most inflamed points, but gradually increasing in quantity with the extension of the inflammation, forms a continuous layer of various thickness and extent. At first it is of the consistence of mucus or thin cream, of a pale yellowish-white or grey colour, and gradually acquires an increase of consistence amounting to that of coagulated albumen or pure fibrine. It is always most consistent the nearer it is examined to the surface of the pleura, the serum which it contains, when first effused, being expelled by its power of spontaneously coagulating, and therefore is most abundant in the more recent deposit, or is collected in the cavity of the pleura. Although presenting sometimes externally a cribriform or rather reticulated appearance, it appears to possess a fine cellular structure; for when it is not firmer than jelly, it may be suspended between the fingers without much of the serum with which it is then loaded making its escape; and when this fluid is forced out by compressing a quantity of the recent lymph in the hand, it resembles a mass of cobwebs moistened with water. It is also important to observe, that the quantity of the effused lymph which becomes organized varies considerably, probably owing to two circumstances, viz. a deficiency or total absence of vital power in the lymph, or the distance at which it is placed from the inflamed surface. The unorganizable part of the effusion is frequently to be recognized by its opaque, granular, or even caseous aspect, and may, in fact, consist either of a puriform or tuberculous secretion, or of both. The other part of the effusion, which, besides the serum, does not become organized, may consist chiefly of plastic lymph, but being separated from the inflamed surface by consolidated layers of the same substance, floats loosely in shreds or flakes in the serum, or being consolidated into masses, in virtue of its contractile property, which necessarily expels from its fine cellular texture the serosity which it contains, falls by its greater

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specific gravity to the most depending parts. It is owing to these circumstances that we find the depending parts of serous cavities, and the spaces formed between the folds of serous membranes, as between the convolutions of the intestines, &c., to contain, when the effusion is general or copious, by far the greatest quantity of solid lymph. In these situations, also, the effusion is most frequently turbid from the presence of a greater quantity of the opaque, unorganizable deposit. It is a curious and interesting fact, that it is in these two opposite conditions of the effusion that the tuberculous affections of serous membranes originate, the mode of formation of which I have endeavoured to explain in the Fasciculus devoted to the consideration of Tuberculous Formations. Whether the effused lymph is separated from the blood, and deposited as an unorganizable product, or whether it is reduced to an amorphous state in consequence of the unfavourable external conditions in which it is afterwards placed, and to which I have already alluded, are points which I shall not investigate at present. It is, however, important to observe, that the individual existence of these two products becomes often conspicuous after the acute inflammation has disappeared, by the cellulo-vascular organization of the one, and the amorphous state of the other, most frequently in the form of small round bodies, or tubercles, connected with the serous membrane. The existence of round or ovoid bodies of a much larger size, sometimes as large as a cherry or pigeon's egg, lying loose in the cavity of the pleura or peritoneum, is a rare but interesting example of the mode of formation and nature of tubercles in the strictly pathological acceptation of this term. In this situation, as in the former, they are composed of two substances, of a pretty dense laminated tissue, frequently consisting of concentric layers, and a nucleus of caseous, putty-looking or cretaceous matter, thus presenting also a striking analogy with the physical characters and mode of formation of phlebolites in the fibrine of the blood, a mode of formation to which I alluded when describing these and other products of a similar origin, and which manifests itself by the eccentric or peripheral disposition of the spontaneously coagulable element of the blood. I have mentioned that detached masses of plastic lymph form in the effusion of inflamed serous cavities, and I have no doubt that the round bodies which I have described as being sometimes formed in these cavities, long after the inflammation has disappeared, have their origin in these masses, and are produced by the same process, an opinion which I am gratified to find is entertained by my friend Dr. Hodgkin, and announced in his valuable work just published on "The Morbid Anatomy of Serous Membranes."

Sometimes at a very early period, at others not until after several days, blood-vessels make their appearance in the plastic lymph of serous membranes. At a very early period of their formation, they are often so numerous as to give an almost uniform red colour to the false membrane in which they are distributed, and are always more numerous at this than at any subsequent period. They diminish in number and size as the false membrane acquires a more perfect organization, and scarcely a trace of them sometimes remains when it has assumed the structure of cellular and serous tissue. Their arrangement, and the manner in which they are connected with the capillaries of the contiguous tissues, I have not been able satisfactorily to ascertain, unless in false membranes extending from one serous surface to another, as between the two pleuræ, or between the opposite surfaces of two folds of the intestines. They are then distinctly seen to consist, each of a flexuous or tortuous trunk, the opposite extremities of which divide and subdivide into numerous minute branches, like the hepatic and abdominal divisions of the vena portæ, which communicate with, and can be easily injected from,

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the general vascular system. This mode of arrangement of the new vessels, which has also been observed by Meckel and Béclard apparently under similar circumstances, is well seen in Plate III. *fig. 5.*

Various opinions are entertained regarding the origin and mode of formation of these vessels. Some pathologists maintain that they are developed under the immediate influence of, and are continuous from their commencement with, those of the inflamed part. Others believe that they are originally formed in the plastic lymph, in nearly the same manner as those which appear in the incubated egg, and afterwards communicate with the capillaries of the general circulation. The former, amongst whom Gendrin occupies a conspicuous place, describe them as being formed by the blood of the inflamed capillaries, which is thrown into the plastic lymph; this, having coagulated, is penetrated by that which follows, and this process being repeated, the rudimentary vessel is prolonged and completed. The latter represent them as being formed by the blood which is generated in the plastic lymph in the form of points, specks, or irregular striæ. According to Laennec, these specks and striæ of blood, which are the rudiments of the future vessels, soon assume a cylindrical shape and a ramiform distribution, by means of which they become connected with the capillary circulation of the neighbouring parts. Their parietes are said to be formed of the fibrine of the blood of the striæ, which, concreting into a rounded substance permeable at its centre, forms a small delicate tube or bloodvessel. From the minute investigations of Gruithausen, Dœliger, and Kaltenbrunner, on this process as observed in the development of the embryo, and the organization of plastic lymph, the following are the phenomena by which it is generally accompanied. The first trace of the new bloodvessel appears in the form of a spot, which soon assumes a more defined and circumscribed shape. In this, granules make their appearance, which present an obscure motion, and the canal in which they are contained becomes elongated and assumes a crescentic form, the cornua of which are turned towards the extremities of the old vessels. The motion of the granules becomes more distinct as the channel in which they move increases in length. The cornua at last reach the extremities of the old vessels, whence the blood is transmitted through the former, and a vascular connection thus established by a kind of loop, between a neighbouring artery and vein. It is in this manner that a connection is established between the new vessels formed in the plastic lymph on the surface of a wound, and the vessels of the general circulation on the same side, but it does not appear to have been ascertained in what manner it is accomplished between the new vessels of both sides, after complete union of the wound has taken place. A similar mode of formation of new vessels has been observed to take place in the plastic lymph which has been effused into the parenchyma of organs, and on the surface of sores during the process of granulation. In the latter situation the lymph deposited on the surface of the sore presents numerous stellated red points, which undergo changes similar to those observed in the lymph of inflamed serous membranes, that is to say, they appear to give rise to the formation of new vessels, which afterwards become connected with the original vessels. The former are prolonged, at isolated points, in the lymph, and produce the vascular projections called granulations. The structure of these was first demonstrated by Professor Thomson of Edinburgh, who showed, by means of injections, that the vessels which they contain, proceeding from the vascular basis of the sore, arrive at their extremities, and there unite in the form of loops. Dr. Allen Thomson, in his interesting and valuable Essay on the "Formation of New Vessels," describes the

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vessels in an injected specimen of granulations, given to him by Dr. Pockels of Brunswick, as having all the form of loops at their distal extremities. In several of the larger granulations, a principal vessel, dividing into smaller branches, is easily seen; the smaller branches formed by the subdivision of one of these principal vessels, unite at their extremities, or pass over, in the form of loops, into those of a larger trunk, thus shewing that these two large vessels bear to one another the relation of artery and vein, while their smaller twigs may be regarded as true capillaries.

From the very general outline which I have given of this interesting process, I am afraid that I have not done justice to the views which it was my object to illustrate; but as it may be considered to partake more of a physiological than of a pathological character, I shall not encroach further on the space which is left for the remaining part of our subject. It may, however, be stated, in conclusion, that the formation of new vessels and the organization of plastic lymph, whether in the parenchyma of organs, on the surfaces of solutions of continuity, or on the free surfaces of serous membranes, is accomplished in one or both of the two following ways: 1st, Under the influence of the *vis à tergo* of the heart and arteries, by means of which the blood is impelled from the original vessels into the plastic lymph, in which passages or canals are hollowed out, which are afterwards converted into bloodvessels; 2d, Under the influence of a *vital agent*, by means of which canals, at first unconnected with the original vessels, are generated in the same substance, which gradually assume the form and distribution of bloodvessels.

Erectile Tissue.—This tissue, which not unfrequently forms in situations in which it does not naturally exist, more especially in the skin of the lips and face, on other parts of the surface of the body, and occasionally in the viscera, has been so denominated from the resemblance of its anatomical characters to those of the natural erectile tissue. It indeed presents varieties similar to those observed in the latter; sometimes consisting of a spongy or cellular structure, intercepted by fibrous tissue, like that of the spleen or corpora cavernosa penis, as when it occurs in the liver; at other times, and indeed by far most frequently, presenting an almost inextricable network of arteries and veins, sometimes the one, sometimes the other of these sets of vessels predominating. The form which this tissue presents is very various. In the liver, the only viscus in which I have seen it, it exists in the form of a round tumour, varying from the size of a large pea to that of a small orange. In the skin and subcutaneous cellular tissue, either of which it may affect separately, but most frequently first occupies the former and extends to the latter, it forms a flat elevation of various shapes, at first, perhaps, not larger than a split pea, a sixpence, or half-crown piece, which may not increase much in its superficial dimensions, or extend over the greater part of the face, neck, and chest. When this is the case, it generally presents a tuberiform aspect, a number of tumours of the size of hemp-seed, peas, or small cherries, projecting from numerous points of its surface, either single or in groups. In one case of this kind, in which I had an opportunity of examining these tumours after death, in an infant, they were formed of the dilated extremities of the vessels, some of which were bulbous and sacculated, and distended with fluid or coagulated blood. One of them burst, and occasioned fatal hæmorrhage. The colour of the skin of the affected part varies from light to dark purple or deep red, and although sometimes of a light red at the commencement, it becomes purple, or all these colours may vary alternately at different periods of the growth of the tumours, and under the influence of changes taking place

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in the general circulation from a variety of causes. The circulation of the blood through the vessels of this tissue, as well as the quantity of this fluid which it contains, does not appear to be sensibly affected by the erectile property ascribed to it. Although it is said to form after birth from accidental causes, I have not seen examples of it except in the lips and at the margin of the anus.

Cellular Tissue.—This tissue, the development of which has already been described, is met with in its most perfect state on the free surface of serous membranes. Although occurring not unfrequently in a membranous form, attached to and covering a portion or the whole of both pleuræ, for example, it is much more commonly met with in the shape of bands of various length and breadth, stretching across the cavity in which it is contained. In both cases it constitutes what are called *adhesions*. In the former case, the opposite surfaces of the serous membrane are united, and the cavity which they formed in the healthy state, obliterated by the intervening cellular membrane, as not unfrequently happens after pleurisy, peritonitis, pericarditis, and inflammation of the tunica vaginalis testis, induced with this view, in the radical cure of hydrocele. In the second case, the opposite serous surfaces are connected at two or more points and throughout a greater or less extent by adhesions of various length and breadth, and do not, as in the former case, interfere mechanically with the change of bulk or place of moveable organs, unless under certain circumstances, as when a lung is bound down to the spine or posterior part of the chest, and the fluid by which it had been compressed is absorbed, it is prevented from following the expansion of the ribs, and consequently from regaining its former dimensions; or, as when the intestines are attached to the anterior parietes of the abdomen, to remote portions of each other, or to the uterus, by strong bands of this tissue, their motion or change of place is impeded, or they are compressed or strangulated. The adhesions which form between the uterus, Fallopian tubes, and ovaries, and the surrounding parts, are much more productive of serious effects than in any other region of the body, and in order to give additional importance to the study of them, I may observe that they are a not unfrequent, and certainly one of the most obvious causes of sterility. They produce, according to their situation and mode of attachment, either anteversion or retroversion of the uterus; they fix the Fallopian tubes in situations in which the fimbriated extremities cannot reach the ovaries; or they envelope the fimbriated extremities in such a manner as to render them quite impervious, (which is always the cause of dropsy of these tubes); or, lastly, they cover the ovaries so completely that impregnation is rendered impossible. See Plate III. *figs.* 6 and 7.

The accidental cellular tissue, whenever it presents a free surface, is always covered by a serous membrane of new formation; and hence arises a frequent source of error regarding the seat of some morbid products. When these products are contained in the accidental cellular tissue, and, consequently, are covered by the new serous membrane, they are often described as being situated on the outside of the original serous membrane, as, for example, in the subpleural or subperitoneal cellular tissue; and this error is the more likely to be committed when the original serous membrane has nearly or altogether disappeared in the situation of cellular adhesions. This is often observed in extensive cellular adhesions of the pleura, peritoneum, and pericardium; and so complete is this change in some cases of chronic pericarditis, that hardly a trace even of the fibrous layer of the pericardium is left, a circumstance which, as Lobstein observes, has given rise to erroneous statements regarding the absence of this sac.

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I have already stated that the bloodvessels at first so conspicuously seen in the recently formed cellular tissue, gradually diminish in size and number, and ultimately disappear. Under these circumstances atrophy supervenes, or the tissues are said to have been absorbed. Although this occurrence is not sufficient to prove the existence of absorbents in accidental cellular tissue, S. V. der Kolk is said to have injected them with mercury.

Adipose Tissue.—This tissue has rarely been met with in any other situations of the body than those in which the natural adipose tissue exists. Andral once found it in the form of a small tumour in the submucous cellular tissue of the intestines, and Laennec in accidental cellular tissue of the pleura. When it occurs in the form of *lipoma*, it is to be regarded rather as the consequence of an excess of nutrition, than as an accidental formation.

Serous Tissue.—I have already adverted to the formation of this tissue, which may be said to be nearly as frequent in its occurrence as the adventitious cellular tissue. We have seen that it takes the form of the cellular membranes and bands which it accompanies, and that it not unfrequently assumes that of the organs which it covers. But we have now to consider it in a more definite and circumscribed form, viz. that of *cyst*, or an organ in most respects similar to the natural serous membranes, deriving, like them, the materials of its nutrition and growth from the surrounding tissues; subject to similar diseases, and exercising in a similar manner the functions of secretion and absorption.

Various kinds of circumscribed shut sacs have been described by pathologists under the denomination of cysts; but under the present head we can only include those which consist of an *adventitious serous membrane*. I may, however, briefly notice the other kinds of cysts, that the distinction between them and the former may become more apparent. Some of these are formed out of pre-existing serous tissues, as in the ovaries in enlargement of the vesicles of De Graeff; in the subcutaneous synovial bursæ and ganglia of tendons, sometimes forming serous cysts of large size; over the patella, olecranon, acromion, &c. the parts most submitted to pressure and friction, as they occur in persons who are often obliged to remain long at work on their knees; and on the shoulders of porters; and they are produced by similar causes after amputation of the thigh at the extremity of the stump. Globular dilatation of the lymphatics, of small veins or arteries (which is extremely doubtful), when obstructed, is said to give rise to the formation of cysts of this kind.

Other cysts are formed out of obstructed ducts and follicles, and are consequently lined by a mucous membrane. Such is the origin of some cysts of the mammæ, which consist of dilated lactiferous ducts, and of those occurring beneath the skin in various parts of the body, formed of distended sebaceous follicles.

The adventitious serous cyst, properly so called, consists of a serous membrane, having the form of a shut sac, the external surface of which has a cellulo-vascular connection with the contiguous tissues, the internal or free surface being in contact with the substances which it encloses. This connection may be formed between it and the natural cellular tissue of organs; or between accidental cellular, fibrous, cartilaginous, and osseous tissues, all of which not rarely contribute, particularly in the liver, to form the entire walls of the cyst. Or, lastly, it may be connected with the heterologous formations, especially the carcinomatous, when they exist as pediculated tumours, and then constitutes their capsular covering, in the form of a reflected membrane, extending over their whole surface to the point of their attachment. It is from this circumstance,

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I believe, that such tumours have been described not only as being contained within, but as being produced by, this kind of serous cyst, the anatomical and physical characters of which have been minutely described by Dr. Hodgkin, and to which he attributes peculiar properties, the exercise of which, he believes, gives origin to many or most of the heterologous formations.

Although the origin of adventitious serous cysts is, in a great many cases, involved in much obscurity, there are good grounds for believing that it is similar in kind to that of adventitious serous membranes, that is to say, that these cysts originate under the influence of causes which excite inflammation of the cellular tissue of circumscribed portions of organs. This is obviously the case when a bullet or any other kind of foreign substance becomes inclosed in a serous cyst. The plastic lymph which is effused in consequence of the inflammation induced by the foreign substance, becomes organized, and is ultimately converted into a serous cyst, probably owing to mere physical causes, such as the pressure of its contents, and the almost universal tendency of membranous tissues, when closed, or prevented from communicating with the exterior, to assume the serous character. Of the same kind are those serous cysts which form around tuberculous matter when it becomes isolated from the surrounding tissues during the process of cure, and those which enclose various kinds of entozoa, as the *acephalocyst*, *cysticercus cellulosæ*, &c. The formation of cysts in the brain after apoplexy, and probably in some cases of circumscribed softening of the cerebral substance, which I have described in the *Fasciculus on Hæmorrhage*, furnishes strong evidence in favour of the opinion that adventitious serous cysts originate in the plastic lymph or fibrine of the blood. In whatever manner, however, the adventitious serous cysts may have been originally formed, they are afterwards variously affected by disease, which gives rise to great variety in the substances which they contain. The ovaries, testis, and *mammæ* are far more frequently the seat of adventitious serous cysts than any other organ of the body. They are also frequent in the kidneys of old persons; but very seldom have they been seen in the brain or lungs. In the organs of reproduction they are generally collected together in clusters; in the brain and lungs solitary; and in the kidneys in both forms, but more often solitary.

The contents of adventitious serous cysts present, as already stated, great variety. Independently of the heterologous formations to which I have alluded, they contain various fluid products; most frequently serosity variously coloured; sometimes an albuminous, mucous, or puriform fluid, at others plastic lymph, fibrine, or blood variously modified and combined, the latter occasionally appearing like chocolate, treacle, or tar, and in great quantity; and lastly, tuberculous matter, cholesterine, and some other anomalous products.

Although these cysts when inflamed present all the physical characters of inflamed serous membranes, the presence of several of the substances which I have named cannot be explained, either with reference to this pathological state or the differences observed in the physical or anatomical characters of the cysts themselves.

Mucous Tissue.—The accidental formation of mucous tissue is observed, 1st, in situations in which the natural mucous membrane does not exist, as in those solutions of continuity which follow inflammation, ulceration, or mortification of the cellular tissue, and which afterwards become the seat of chronic abscess, whether in the form of a shut cavity, or communicating with the external surface of the body by what is called a *fistulous passage*; 2d, in the situation of a natural mucous membrane which has been

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destroyed, the cicatrix which supplies its place consisting of a mucous tissue. The formation of mucous tissue in the former situations, and its reproduction, as it is called, in the latter, has been studied with considerable care by several pathologists, since its existence was first made known by John Hunter, as an essential element of the suppurating surface of fistulous communications. The following are the most important of the circumstances connected with the formation and anatomical and physiological characters of this tissue.

Whether we consider the mucous tissue as forming a cicatrix, or the lining membrane of an abscess or fistulous passage, it originates in the organization of the plastic lymph which is deposited in the inflamed cellular tissue. In the first stage of the process, the cellular tissue is consolidated by the lymph, of which there is also a layer on its denuded surface; it then acquires a vascular structure, which often produces a deep red colour, accompanied by an irregular granular appearance in this situation, and here also it afterwards gradually assumes either a uniform, smooth, glossy, or rough villous aspect, or that of simple mucous membrane. The mucous tissue thus formed adheres intimately to, or is confounded with, the cellular tissue, which has either regained its natural state or has acquired a considerable degree of density. The colour which it presents varies much in abscesses and fistulæ, according to the degree of inflammation, the quantity of blood which it contains, and the chemical action of the gaseous products to which this fluid is often subjected. It is generally, however, of a reddish or greyish blue colour in these situations, and in the intestines differs so little from that of the natural mucous membrane, that the cicatrix which it forms would escape detection if not carefully sought for. See Plate IV. *fig. 4.*

In no situation in which the accidental mucous tissue is found, does it present any trace of muciparous follicles or glands, and the villi which project from its internal surface, both as regards their form and structure, differ from those of natural mucous membrane. It is therefore to be regarded as a simple mucous membrane, analogous to that of excretory ducts. It performs the functions of secretion and absorption, thereby modifying greatly the quantity and quality of its contents. In the absence of suppuration and irritation, it secretes, particularly in fistulæ, a mucous fluid, which, according to Andral, sometimes resembles the natural mucus, both in its physical and chemical qualities. Villermé states that he has found the mucous lining of these fistulæ covered by an epithelium continuous with the epidermis. The accidental, like the natural mucous membrane, does not unite when inflamed, as the secretion which it furnishes is not susceptible of a vascular organization. Hence the necessity of destroying it in order to effect the obliteration of fistulæ and many chronic abscesses.

Cutaneous Tissue.—Cicatrices of the skin afford frequent opportunity of observing the accidental formation or reproduction of this tissue. Having already described the organization and mode of formation of the granulations which arise in the plastic lymph deposited on the surface of a sore, I have now only to advert to the changes which these undergo during the development of the new skin. The red colour of the granulations diminishes insensibly; these bodies themselves diminish in bulk, become less prominent, and approximate more closely to each other at their basis; and if their vascular structure is now examined, it is found to be much less apparent than before, the vessels being much smaller and less numerous. The first appearance of the new skin is generally seen along the margin of the old skin, but is also sometimes observed in isolated points on the surface of the sore, particularly when this is of large size. The new skin is

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recognized by the presence of a thin, pearly-coloured film, which, as it extends, acquires greater thickness and density, changes which are effected on the surface of the granulations from without inwards, by the superficial portion of these acquiring, in consequence of the contraction and obliteration of their vessels, a cellulo-fibrous structure. In this state the cicatrix has a very imperfect resemblance to the old skin; it is more dense and compact, is confounded with the subjacent cellular tissue, and is not separable into cutis, rete mucosum, and epidermis. After a time, however, we find in it these elements of the cutaneous tissue in a rudimentary state; the epidermis can be separated from the cutis, and the presence of the rete mucosum has been inferred from the colour of the cicatrix, although it is, in general, but a faint resemblance of, and often very different from, that of the surrounding skin. Although the papillæ are sometimes partially developed in the new skin, the muciparous glands and the hair do not appear to be reproduced if the cicatrix succeeds to the entire destruction of the cutis.

Hair.—The accidental development of hair is seen to take place most frequently on those parts of the body affected with nævi, where it is often very abundant, fine or coarse, short or long, and of various colours. But here, like the nævi which it accompanies, it is to be considered rather as a congenital than an accidental formation. So also is the hair so often found in ovarian cysts. That found in subcutaneous cysts, as those of the scalp and eyelids, is probably the result of accidental development, as it arises from the interior of these cysts, which is lined by the reflected cutaneous tissue. The most remarkable examples of the accidental development of hair are met with in some portions of the natural mucous membrane, as that covering the caruncula lachrymalis, the tongue, and urinary bladder. In the latter situation, however, it has always been found loose, without bulbs, seldom more than an inch in length, and has generally been passed in the urine with the phosphate of lime, sometimes in great quantity.

Nails.—The reproduction of the nails, and the development of a horny tissue on the external surface of the body, occurs under a variety of circumstances. When a nail has been destroyed or removed, it is reproduced by the formation of a horny substance, from the root outwards, after the manner of the original nail. It sometimes differs very little from that which it replaces; at other times it is deformed, thicker and shorter, or longer and hooked, and frequently split or separated into irregular portions at the extremity.

An analogous or *horny tissue*, as it is called, is occasionally formed in the situation of cicatrices and some ulcers, and consists of concrete albumen, furnished by the inflamed cutis, which, as it is deposited, acquires a considerable degree of hardness, and sometimes assumes a conical or spiral arrangement. It occurs most frequently in the cicatrix of an amputated finger, sometimes on the scalp and other parts of the body, in the situation of tumours or ulcers. The *ichthyose cornée* of Alibert, consisting of a morbid secretion of the cutis, occupying the situation of the epidermis, appears to be a modification, chiefly in form, of the preceding.

Fibrous, Fibro-cartilaginous, Cartilaginous, and Osseous Tissues.—As these analogous tissues are observed to occur under nearly similar circumstances, and frequently succeed each other in the order in which I have enumerated them, I shall include them all under the same general description. The series of changes which take place in the cellulo-vascular tissue of organized lymph, by means of which these tissues are produced in succession, are very similar to those which are known to accompany their development

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in the early and subsequent periods of foetal life. The cellulo-vascular always precedes the fibrous, this the fibro-cartilaginous, and the cartilaginous the osseous tissue. It is in this order that we find these tissues forming and constituting, under a variety of forms and of various extent, the external parietes of cysts; lining serous membranes, as the pleura, peritoneum, tunica vaginalis testis, &c.; and forming sometimes a part or the entire of the adhesions which unite the free surfaces of organs, as those between the pericardium and heart, the diaphragm and liver, and the opposite surfaces of joints. In the same order also do they appear in the reunion of fractures and the reproduction of bone, and as it is here that the study of them and the purposes which they serve are most interesting, because most important, I shall give a general outline of the process by means of which their formation is accomplished.

Whether the formation of new bone is to effect the reunion of a fracture, or the restoration of a loss of substance of the old bone, the process by means of which it is accomplished is essentially the same as that which takes place in lesions of a similar kind of the soft parts. Inflammation, the effusion of plastic lymph, the cellulo-vascular organization of this, and its gradual conversion into fibrous, fibro-cartilaginous, cartilaginous, and osseous tissues, follow each other in succession. The material out of which the new bone is formed is the plastic lymph, which is furnished by the periosteum, medullary membrane, surrounding cellular tissue, and the substance of the bone itself, according as the inflammation by which it is preceded affects one or all of these parts, a circumstance which will depend chiefly on the original seat or extent of the injury or disease. The parts which contribute most to the formation of the new bone, and in which this process is first established, are those whose anatomical and physiological conditions render them most susceptible of inflammation, or in other words, those whose vital endowments are greatest, and which, consequently, furnish the most abundant supply of plastic lymph. These are, 1st, the periosteum; 2d, the surrounding cellular tissue; 3d, the medullary membrane; 4th, the substance of the bone. It is not until some time after the three former have been the seat of inflammation, and the plastic lymph effused by them has become organized, that the substance of the bone participates in the same change, and takes a part in the regenerative process. The plastic lymph is always furnished in greatest abundance by the periosteum and surrounding cellular tissue, as is clearly shown by the appearances which accompany fracture of a long bone. It is found much more abundant around the circumference, and in the situation of the fracture, than elsewhere; and if the inflammation has been severe, forms a mass of considerable extent projecting outwards, and dipping between the fractured extremities, where it meets with that effused by the medullary membrane. Hence, also, the cellulo-vascular organization of the lymph, and the subsequent changes which it undergoes, are effected in the same relative order, the formation of the new tissues being far advanced or completed around the circumference of the bone, before it has commenced or has made much progress between the fractured extremities, and especially between these and the uniting medium. It is on account of the ossific process being first completed in the tissues around the bone, that these are said to form the *provisory callus*, a provision, indeed, which affords time for the development of a vascular connection between them and the extremities of the bone, and the accomplishment of a permanent bony union.

Although the formation of these several tissues takes place in the order in which I have stated, it must be observed that the process of reproduction may be arrested in one or other of its successive stages, that is to say, a fibrous or fibro-cartilaginous instead

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of an osseous tissue may form the uniting medium of a fracture, or supply the place of a loss of substance of the old bone. This is seen, in general, to follow caries or necrosis of the flat bones, or of the external parietes of the long bones with destruction of the periosteum; resection of a part of the body or of the extremities of these; fracture of the neck of the femur, of the olecranon, and of the patella; and is explained on the principle already alluded to, viz.—a difference in degree of the vital endowments possessed by the tissues concerned in the reproductive process. In such cases, union or reproduction is imperfect, and is effected either by the fibrous or fibro-cartilaginous tissue. It is also to be observed that the osseous tissue, even in cases of the most perfect bony union, may be the result of the conversion of the fibro-cartilaginous rather than of the cartilaginous, into the osseous tissue, and that this latter often acquires a degree of hardness which is much greater than that of the original bone.

As the reproductive process never appears so effective as in the case of necrosis of the entire shaft of a long bone, I may, although it is here essentially the same as in reunion of a fracture, give an outline of the successive stages as observed in necrosis of a long bone in an animal, produced by the introduction of a foreign body into the medullary canal. The periosteum and surrounding cellular tissue become inflamed over the whole surface of the bone; great congestion, the effusion of serosity, lymph, and pus succeed, and produce considerable swelling of the limb. After some time fistulous communications are established between the interior of the old bone and the external surface of the limb. At this period the presence of a new bone, containing the old one, is ascertained by the introduction of a probe through the fistulæ, and by means of which we can perceive that the latter is detached or moveable throughout the whole or the greater part of its length. After the discharge or removal of the old bone, the new one by which it is replaced, and which is found to be united with the epiphysis of the former at an early stage of the reproductive process, gradually acquires greater solidity, contracts upon itself, and ultimately approaches to the structure of the original bone. The suppuration which accompanied the presence of the dead bone ceases soon after this has been removed, and the space which it occupied is gradually converted, by the contraction of the new bone, into a medullary canal. In the human subject, when necrosis of a long bone is the consequence of inflammation of the periosteum, of the medullary membrane, or of both, the reproduction of the new bone is accomplished in a similar manner.

As it would be inconsistent with the plan of this work to give a separate description of each of the analogous tissues, I shall conclude the general view which I have given of their mode of formation and development, by a few observations on the fibrous and cartilaginous tissues, in regard to the occurrence of the former as a contractile tissue, and of the latter in the form of isolated round or ovoid bodies in the cavities of serous and synovial membranes. The fibrous tissue, whether originating in the plastic lymph of serous membranes or of that of the cellular tissue of organs, and when it does not undergo any further change towards the formation of the fibro-cartilaginous or osseous tissue, has a greater or less tendency to contract upon itself, and consequently to modify the nutrition, bulk, form, and situation of the parts with which it is connected. Atrophy of a lung, when this organ is covered by a fibrous envelope in chronic pleurisy; stricture of the intestines after cicatrization of ulcers which had destroyed the muscular coat around the circumference of the tube; displacement of the fingers, of the lower lip and jaw, &c. after burns—are effects of the contractile property of this tissue,

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illustrations of which are given in the Fasciculus on Atrophy, and in the present one, Plate IV, *figs.* 5, 6, and 7. The almost irreparable mischief which this tissue in the cicatrices of burns occasions, is a striking example of its contractile power, and which I have known to produce complete closure of the mouth and nostrils, and death from inanition. When treating of the vascular organization of lymph in the form of membranous adhesions and granulations, I stated that these diminish in bulk, and acquire an increase of density in proportion as their vascular element becomes less apparent and they assume the fibrous character. These changes I regard as the early indices of the contractile property which afterwards becomes so apparent in this tissue in the cicatrices of burns, and indeed of most cicatrices which succeed to a considerable loss of substance, more especially when the process of regeneration is accomplished by granulation. And it is also to be observed, that the more exuberant and vascular the granulations, the more certainly is the contractile property acquired, and its effects manifested in the cicatrix which follows. These conditions of the granulations are decidedly much more conspicuous after burns than after any other lesion, and it is on this account, as well as from the facts already noticed, that I feel disposed to ascribe the existence of this property to an excess of the vascular in combination with the fibrous tissue. This hypothesis derives support from the fact that the contractile property of this tissue is slight in degree when the process of granulation is retarded, or its activity repressed, before cicatrization is allowed to take place.

The cartilaginous tissue to which I have alluded is that which occurs in the form of round or ovoid bodies contained in the cavities of synovial membranes, and regarding the origin of which there is much diversity of opinion. I have already described the formation of similar bodies in the concrete plastic lymph of serous membranes, and which are found loose in the cavity of the pleura, peritoneum, and tunica vaginalis testis. Those found in the cavities of synovial membranes have, probably, a similar origin. They are met with most frequently in the knee-joint, and have often been known to succeed to injuries of this part. It is therefore extremely probable that inflammation of the synovial membrane of the joint, terminating in the effusion of plastic lymph, gives rise, in the manner already explained, to the formation of these bodies. It is possible that they may also originate in the manner described by Béclard, that is to say, the cartilaginous formation having taken place in the cellular tissue, behind the synovial membrane, carries before it, in proportion as it increases in bulk, a portion of this membrane, which, having become pediculated, is at last detached, and drops into the cavity of the joint. John Hunter ascribed their origin to the presence of coagula, which, becoming organized, formed the loose cartilaginous bodies. I have not, however, met with any examples of either of these modes of formation of these bodies, but I have found fibrous tumours in the cavity of the abdomen, which had been originally situated beneath the peritoneum covering the fundus and broad ligaments of the uterus, and I once saw a small, pyriform, fibrinous coagulum, covered by a prolongation of the peritoneum, and supplied with several bloodvessels from this membrane, in a rabbit (a case similar to that observed by John Hunter,) which, at some future stage of its development, would no doubt have become detached and fallen into the cavity of the abdomen.

ANALOGOUS TRANSFORMATIONS.—The conversion of existing tissues into others of a different kind constitutes the distinctive character between the analogous transformations and the analogous formations, the latter being superadded to these tissues, or

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replacing them when destroyed. With this difference however, they are subjected to the same laws which preside over the regular development of the natural tissues, and observe the same order or gradation by which these pass the one into the other at different periods of foetal life and in different animals. Thus the cellular tissue is transformed into serous or fibrous tissue, and this into fibro-cartilaginous, cartilaginous, and osseous tissues in succession, constituting, as already observed, the ascending series; and when taking place in the reverse order, the descending series of this class of analogous tissues. Hence it follows that those tissues which, in the physiological condition, do not observe this order of succession, are not found to do so in the pathological condition. Thus bone, muscle, nervous matter, &c. are not successive developments of each other in the physiological condition, and do not occur in the pathological.

All the transformations of the first series may originate in the natural cellular tissue, all those of the second in the other tissues and in organs. They are not equally frequent in all these tissues; those of the first series are most frequent in the cellular and fibrous tissues; those of the second in glandular and parenchymatous organs. These organs, indeed, are susceptible of undergoing only the second series of transformations, as well as the muscular and nervous tissues. The mucous and cutaneous tissues are subject to both, being convertible the one into the other, as also the cartilaginous and osseous, the fibrous and cellular tissues.

The fibrous and osseous transformations, from their greater frequency than the others, as well as the importance of the morbid states to which they give rise, deserve some additional illustration.

The fibrous transformation exists under a variety of forms, but I shall confine myself here to a description of what are called fibrous tumours, which are chiefly composed of this tissue. These tumours originate in the fibrous membranes and the cellular tissue of organs, and vary considerably in bulk and number. They are most frequently found in the parietes of the uterus, where they vary from the size of a pea to that of a child's head, or even larger. Their anatomical and physical characters present considerable variety. Some of them consist of irregular bundles of fibrous tissue, interlacing each other in different directions, connected together by loose cellular tissue; others present a compact structure, composed almost entirely of bundles of fibrous tissue, interwoven together in the form of irregular or rounded masses, or having the appearance of interrupted concentric rings when cut. These appearances are more or less marked according to the quantity of cellular tissue which is present. When collected between the individual masses or smaller tumours, it gives to the entire tumour a lobulated character; and when intermixed with small nodules of the fibrous tissue, produces a granular appearance.

The density of the fibrous tumours varies with the quantity of the cellular tissue which they contain. When this tissue is abundant, they may be crushed or torn into shreds; but when the fibrous tissue predominates or constitutes nearly their whole bulk, they are often so hard that division of them with a knife is not easily accomplished. The colour of the fibrous tissue in these tumours is of a dull or bluish-white, sometimes of a pearly lustre; at others of a light yellow or rose tint. The number of bloodvessels which it receives is always in proportion to the quantity of cellular tissue with which it is connected. The large branches ramify in the cellular tissue, a few only of their minute subdivisions passing between the laminae or bundles of the fibrous tissue.

The fibrous tissue frequently undergoes the fibro-cartilaginous and osseous trans-

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formations; but although it has been maintained, on the authority of Dupuytren and some other pathologists, that it is apt to degenerate into malignant disease, I do not believe I have ever seen a case in confirmation of this opinion.

Although the osseous transformation has been described as occurring in most of the tissues, even in the muscular and nervous tissue, it is exclusively confined to the cellular, fibrous, fibro-cartilaginous, and cartilaginous tissues. Although occasionally met with in some of these tissues at an early period of life, it is generally not until after the age of forty or upwards that it makes its appearance in the cartilaginous tissue, the cellular and fibrous tissues of the vascular system. No probable reason has been assigned for its occurring so frequently in the arteries and so seldom in the veins, except perhaps that the former, from the greatly increased exercise of their function, compared with that of the latter, arrive sooner at that condition which constitutes the period of decay, a condition which in this as well as in other tissues is so favourable to its production. The osseous transformation occurs most frequently in the aorta, the arteries of the brain, and lower extremities; much more seldom in the valves of the right than in those of the left side of the heart; rarely in the pulmonary artery and veins. Its seat in the arteries is the cellular tissue which separates the inner membrane from the middle coat, and in the middle coat itself. It appears in the form of small circular, oval, or irregular patches, of a dull white or yellowish colour, of the consistence of cartilage, in the former; and in that of plates, spiculæ, or rings in the latter, occupying a greater or less extent of the vessel, portions of which are sometimes converted into an osteo-cartilagenous tube resembling the trachea of a bird.

The internal surface of the vessel becomes unequal, its lining membrane is ruptured, fibrinous coagula form around the projecting pieces of bone, and obstruct or arrest the circulation of the blood; or this fluid, escaping through the lacerated coats, gives rise to aneurism or hemorrhage. A less perfect bony formation also takes place in the cellular tissue, between the inner and middle coats, appearing at first like soft plaster, which afterwards concretes into irregular patches; or rupturing the inner membrane, some of it probably escapes into the cavity of the vessel. This constitutes the atheromatous degeneration of the arteries of some pathologists. It is, perhaps, a more frequent cause of rupture and aneurism of the arteries than the former.

The osseous transformation is always very imperfect in the vascular system. It contains a much greater quantity of earthy matter than the natural bony tissue, is therefore much harder, and in general dry and brittle. It rarely presents, in any situation in which it is formed, either medullary cavities or periosteum. Cartilage and fibro-cartilage are perhaps the only tissues in which this occasionally occurs.

It is doubtful whether what has been described under the name of the *fatty transformation* of organs belongs to this division of our subject, as some pathologists consider the alteration to consist in the superaddition of a fatty matter to the natural tissues of organs, rather than in the conversion of these into fat. I am, however, disposed to believe that a transformation of this kind takes place in some organs, as the muscles and liver, and sometimes in the kidneys and pancreas. It is most marked in the muscles of the inferior extremities of old persons affected with paralysis, and which have long remained in a state of absolute rest. In such cases I have found the affected muscles but little altered, except in their colour and chemical composition. They are of a pale-white or straw-colour, diminished rather than increased in bulk, and the arrangement of their fibres perfectly distinct. Indeed, the larger bundles of the

ANALOGOUS TISSUES.

muscular fibres, and their subdivision into smaller ones, can easily be traced with the scalpel. When dissecting the muscular fibres, or when these are pressed between the fingers, a clear oily fluid oozes out, and which, when a portion of the muscle has remained some time in alcohol, collects in considerable quantity on the surface of this spirit. According to Lobstein, the chemical analysis of muscular tissue which has undergone the fatty degeneration affords an oily matter, a substance resembling boiled muscle, gelatine, adipocire, and solid fat. The fatty transformation of the liver is met with in persons, chiefly females, who have died of phthisis. This organ is sometimes much increased in bulk; presents a uniform straw or pale white colour; feels fatty to the touch, and leaves an oily or a greasy stain on the scalpel with which it is cut; it receives readily the impression of the finger, and is broken into fragments under very slight pressure. Vauquelin found the fatty liver composed of forty-five parts of yellow concrete oil, nineteen parts of parenchyma, and thirty-six of serosity.

DESCRIPTION OF THE PLATES.

PLATE I.

N.B. The amorphous formations of inflamed mucous membranes represented by the figures in this plate illustrate the subjects treated of in the preceding Fasciculus. *Fig. 1*, Croup; the false membrane confined to the trachea and larynx. A, tongue; B, B, amygdala; C, C, pharynx; D, D, œsophagus; E, epiglottis; F, F, trachea; G, G, bronchi; H, K, the false membrane occupying the larynx and trachea, detached and broken near the bifurcation of the latter, and portions of it in the bronchi. *Fig. 2*, Croup in the adult, the false membrane occupying the trachea and bronchi to their termination in the air-cells. A, epiglottis; B, C, C, trachea, containing a tubuliform membrane D, in the upper extremity of which a small piece of wood is represented to shew more clearly that it is hollow; its division, at the bifurcation of the trachea E, into branches corresponding with the bronchi, F, F, F, and G, which, at H, are traced nearly to their termination in the air-cells; K, L, ulceration of the epiglottis and trachea. *Figs. 3 and 4*, Pneumonia, with false membranes in the bronchi and air-cells. *Fig. 3*, A, B, represents the tubular membranes of the bronchi, of considerable size, terminating abruptly, C, D, at some distance from the air-cells; E, E, pulmonary tissue, engorged with blood; F, the same, infiltrated with serosity, lymph, and pus. *Fig. 4*, a small portion of hepatized lung; A, B, the cut extremities of tubular false membranes occupying the small bronchi, and air-cells C, D, D. *Fig. 5*, a ramiform membrane of the same kind, which was detached and expectorated with others from time to time. A, A, minute branches corresponding to the last divisions of the bronchi; B, B, others of similar dimensions collected and held together by a little mucus. *Fig. 6*, a portion of the ileum, the mucous membrane of which is covered with a thin layer of pseudo-membrane; in some parts continuous, in others in patches and minute points.

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P L A T E II.

Fig. 1, polypus of the heart. A, right, B, left ventricle; C, right, D, left ventricle; E, F, auriculo-ventricular valves; G, a pyriform polypus attached to the muscoli pectinati of the right auricle; H, a flat polypus attached in the same manner in the left auricle. *Fig. 2*, a section of the pyriform polypus, showing its pediculated attachment, A; its fibrous structure, B; and the cavities, C, D, in its interior. *Fig. 3*, another form of polypus attached to the tricuspid valve. A, right auricle; B, B, muscoli pectinati; C, C, tricuspid valve; D, D, a large granular polypus, surrounding and attached to the tendons of the valve. *Fig. 4*, a section of a portion of the polypus, shewing its attachment to the tendons, A, A, and its fibrous reticulated structure, B. *Fig. 5*, obliteration of the vena cava inferior, iliac, and femoral veins. A, vena cava; B, C, iliac veins, obliterated by a fibrous tissue, D, D, extending to the bifurcation, E, of the cava, from which point it is also obliterated for nearly three inches in length by a firm, fibrinous coagulum, F, attached inferiorly by cellular adhesions, G, G, to the internal parietes of the vein; H, H, H, enlarged veins, by means of which a collateral venous circulation was established between the veins of the lower extremities and the cava. *Fig. 6*, sections of the obliterated femoral veins, shewing the formation of canals in the organized fibrine, by means of which the circulation may be restored. A, a longitudinal section, traversed by an irregular canal containing red blood; B, C, transverse sections containing a laminated fibrous tissue united with the walls of the vessel; D, E, similar sections, shewing the orifices of several canals. *Fig. 7*, transverse sections, A, B, C, of the femoral vein, presenting similar appearances. *Fig. 8*, a portion of the aorta, the lining membrane of which was covered with a false membrane of considerable thickness, containing small bony patches. A, the middle coat, undergoing the osteo-cretaceous transformation; B, the lining membrane, the cellular tissue which separates it from the former undergoing the same change; the false membrane, coloured by imbibition, and containing several bony patches.

P L A T E III.

Fig. 1, phlebolites. A, fundus of the uterus; B, right Fallopian tube; C, trunk of the veins of the broad ligament, several of which contain phlebolites in various stages of formation; D, D, several phlebolites fully formed; E, E, others in the state of coagula. *Fig. 2* represents these bodies removed from the veins; those marked 1, 2, 3, &c. in the upper row are entire, those in the lower row are cut to show their internal arrangement. *Fig. 3* represents a coagulum formed around a thread in the axillary artery of a dog. A, the artery entire, red, and swollen over the coagulum; a, the extremity of the thread; B, the artery laid open, the fusiform coagulum suspended at its middle by the thread, a; the inferior extremity of coagulum, b; C, coagulum laid open, shewing the thread, a, contained within it, and passing down towards the basis, b. *Fig. 4* represents endocarditis and the accidental formation of fibrous tissue in the mitral valve. A, left ventricle; B, aorta; C, left auricle; D, coagulable lymph

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adhering to the lining membrane of the auricle; E, inflammatory injection of the parietes of the auricle; F, F, mitral valve converted into bands of fibrous tissue, with considerable narrowing of the orifice. *Fig. 5*, new-formed vessels in the cellular adhesions uniting the intestines with each other and with the parietes of the abdomen. A, A, a portion of intestine; B, B, a portion of the parietes of the abdomen; C, C, the adhesions in which the bloodvessels present an arrangement resembling that of the portal system. *Fig. 6*, the Fallopian tubes obliterated at their fimbriated extremities by accidental cellular and serous membranes, and the ovaries enclosed in a capsule of the same. A, fundus of uterus; B, B, fimbriated extremities of Fallopian tubes; C, ovaries. *Fig. 7*, extensive cellular adhesions, enveloping the ovaries and obliterating the Fallopian tubes. A, fundus of uterus; B, Fallopian tubes obliterated at C, and attached to the body of the uterus; obliterated also at D, and distended by an accumulation of serosity; E and F, the ovaries, the former nearly concealed by the accidental cellular tissue.

P L A T E I V .

Fig. 1, a small pyriform, organized coagulum, attached by a pediculated extremity to the parietes of the abdomen and covered by a serous membrane, apparently a prolongation of the peritoneum. It was found in a rabbit. A, the tumour; B, B, B, small vessels ramifying under the peritoneum and passing along the pedicle into the tumour. *Fig. 2*, a section of the tumour composed of red-coloured fibrine externally, C, and the same substance in the form of a nucleus, B. *Fig. 3* represents the cartilaginous transformation of the spinal arachnoid of old persons: it extended from the cervical to the termination of the lumbar portion of the cord. A, A, A, dura mater laid open; B, spinal cord; C, C, C, cartilaginous plates of various sizes, situated in the internal surface of the arachnoid, perhaps occupying the substance of this membrane. Their anterior surface was perfectly smooth, their internal rough and spiculated; they were of a pearly tint, flexible, and broke, when bent, like natural cartilage. *Fig. 4*, cicatrization of ulcers of the glandulæ agminatæ, after typhoid fever. A, B, C, glandulæ agminatæ; D, E, cicatrices consisting of simple mucous tissue; F, G, two small ulcers nearly cicatrized, their edges, however, are still sharp; H, another ulcer undergoing the process of cure. *Fig. 5*, imperfect cicatrization of an ulcer of the small intestine which had destroyed the muscular coat around the whole circumference of the tube. It has been replaced by the contractile fibrous tissue, A, which here has a stellated arrangement; and has by its contraction narrowed the intestine, B, B, considerably. *Fig. 6*, a remarkable example of stricture of the small intestine, from the presence of the same tissue in a cicatrix occupying the entire calibre of the intestine. A, upper extremity of the ileum; B, lower extremity; C, C, the fibrous tissue stretching across the intestine in the form of bands, leaving small openings, D, between them, through which only small quantities of liquid fæces could pass. *Fig. 7*, the contractile fibrous tissue of burns, which has produced great distortion and displacement of the lower lip and skin of the neck. A, B, highly vascular bands of this tissue, indicating its formation and extension; C, C, C, smaller bands of various degrees of vascularity, and possessing more or less of the contractile property of this tissue.

Plate I.

Fig. 6.



Plate I.

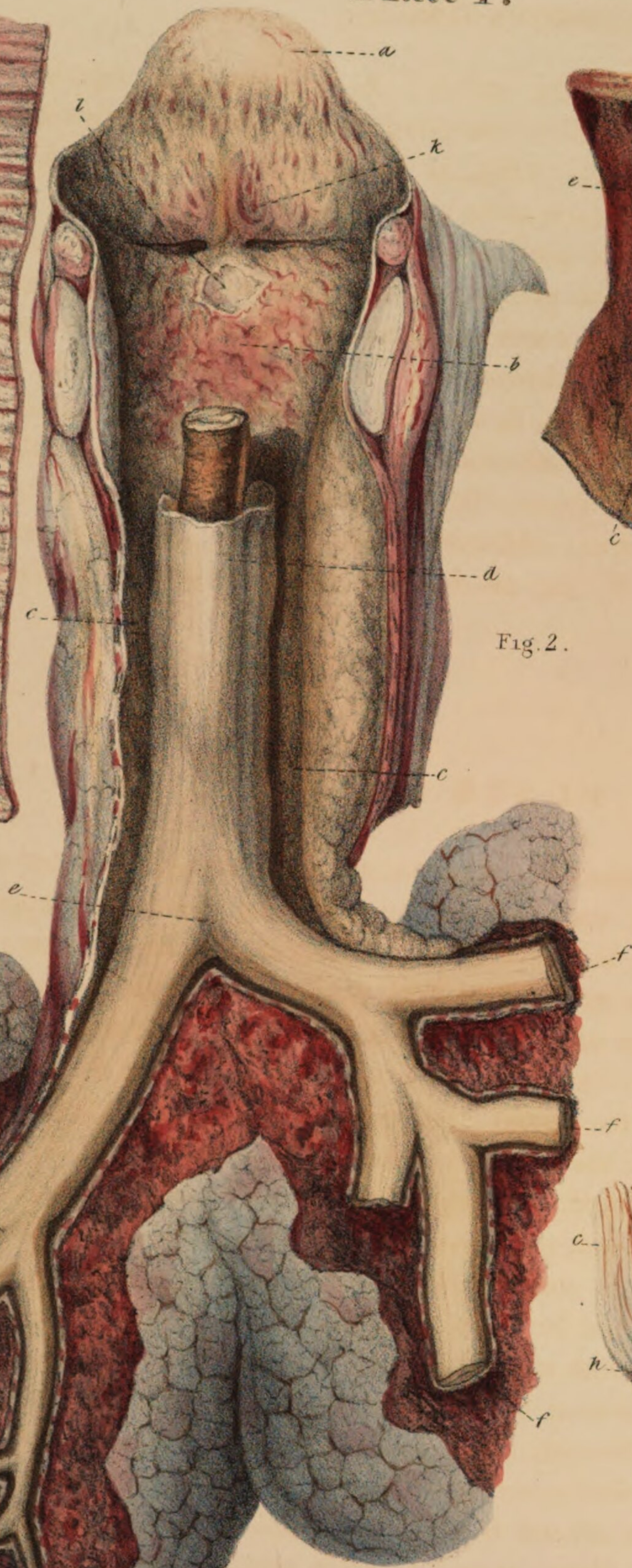


Fig. 2.

Fig. 3.

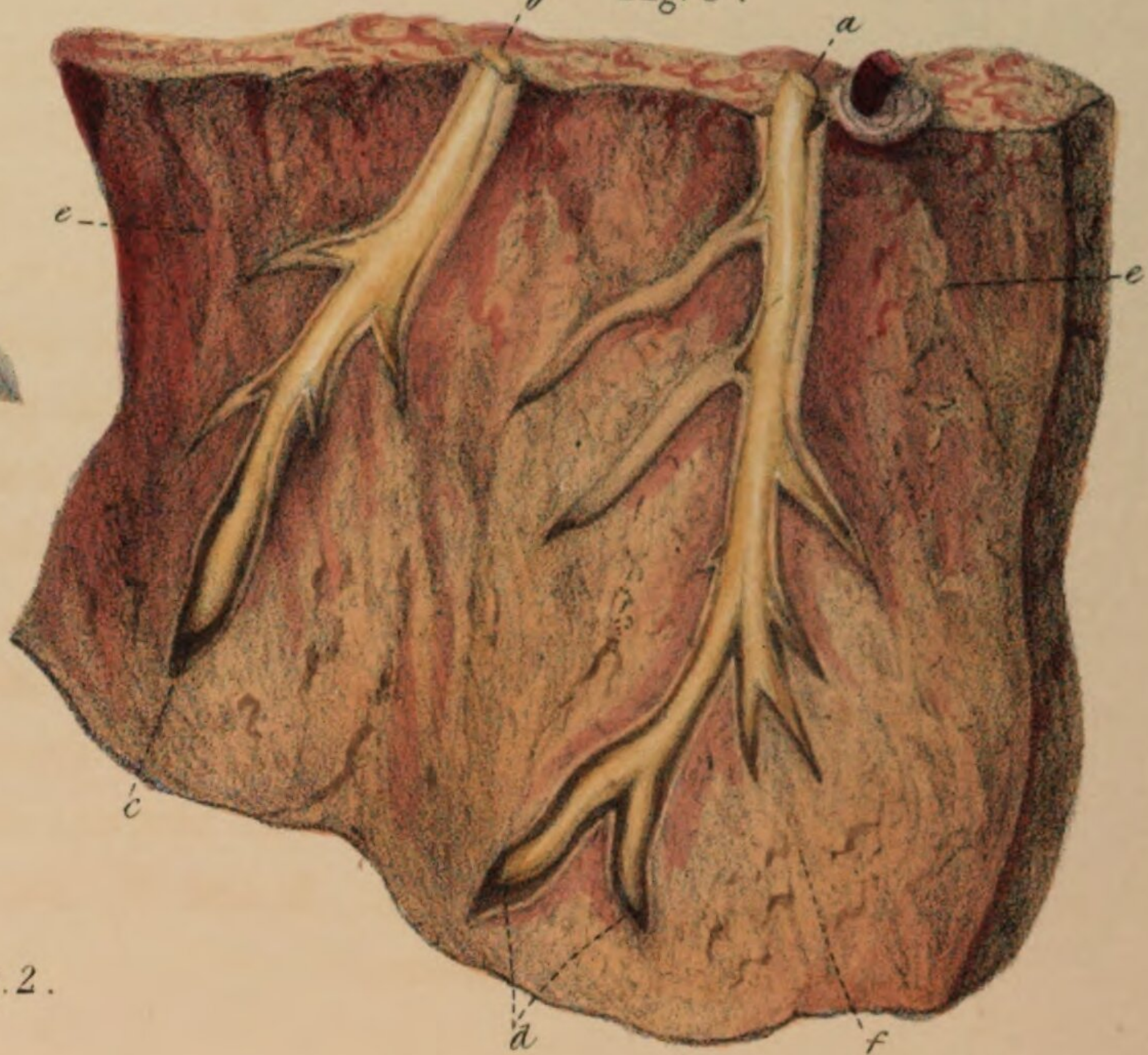


Fig. 1.

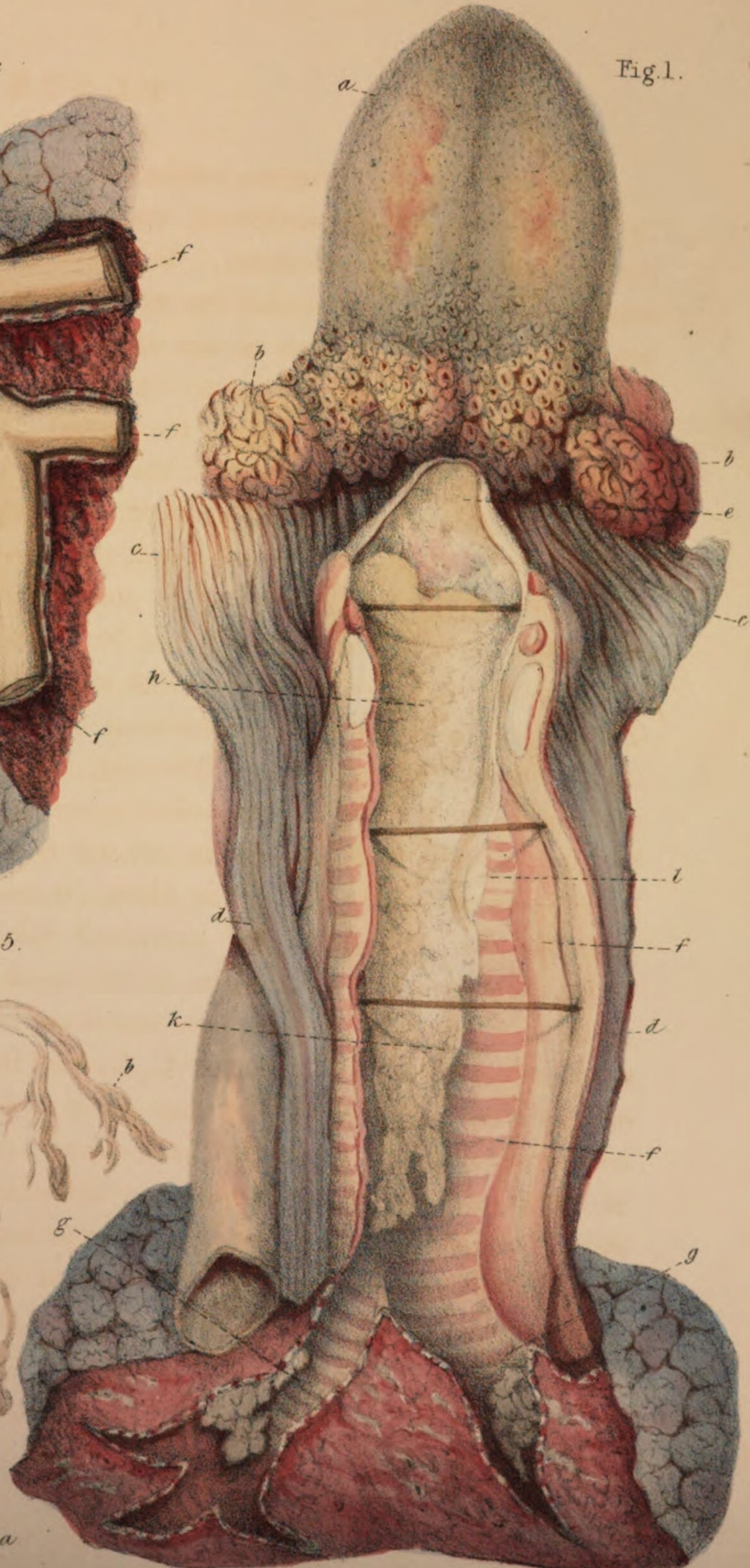


Fig. 5.



Fig. 4.

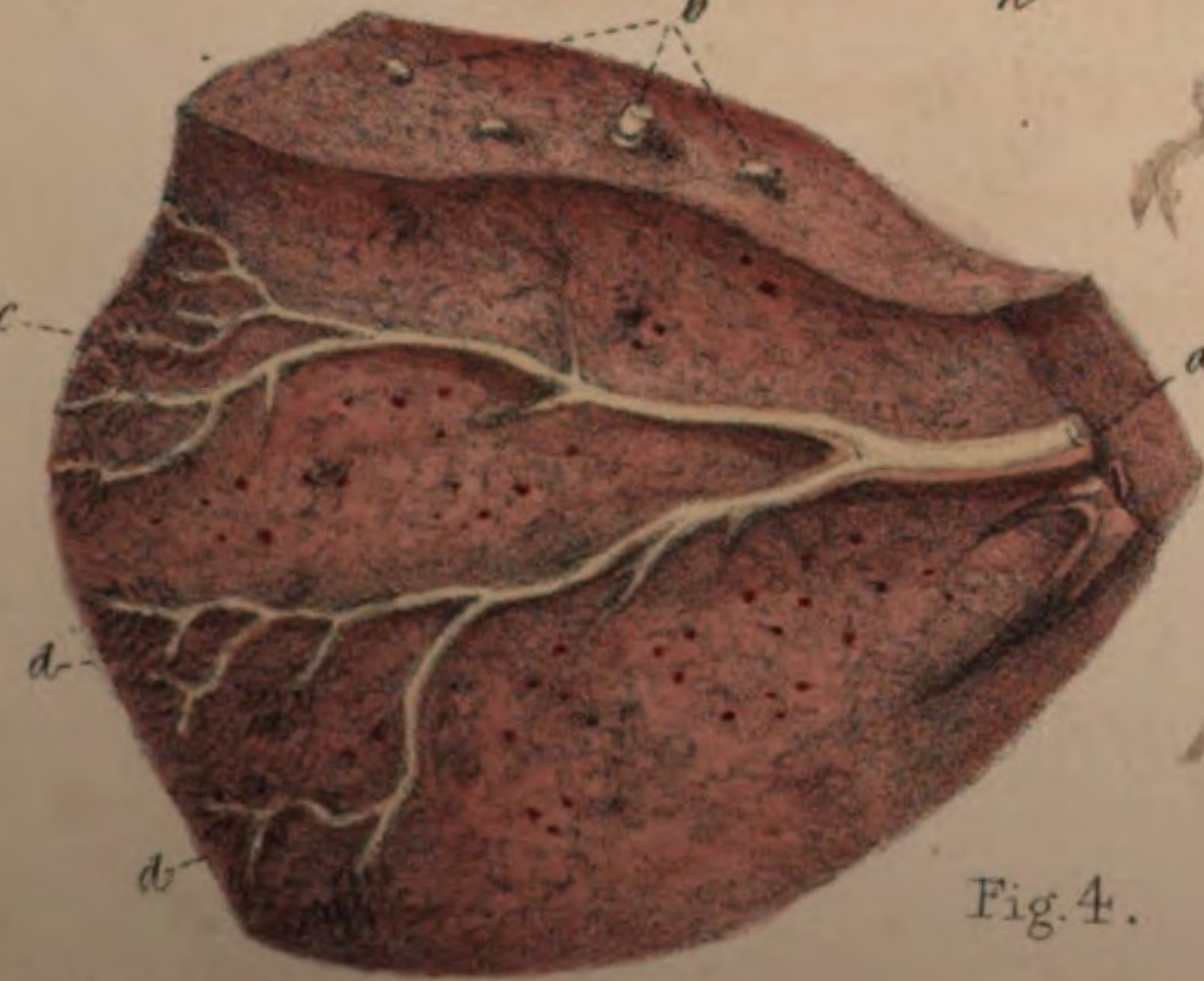


Plate II.

Fig. 1.

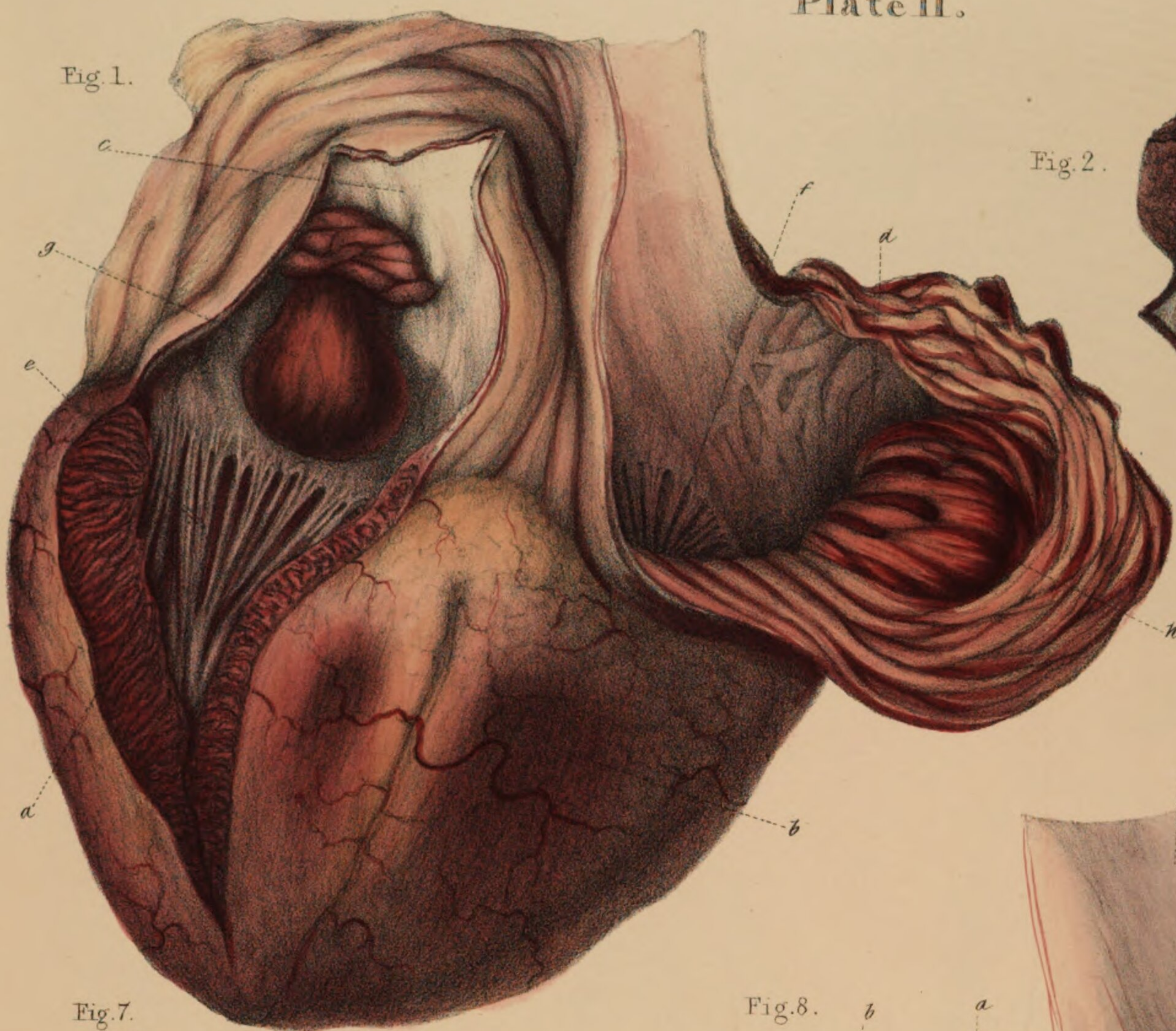


Fig. 2.



Fig. 7.

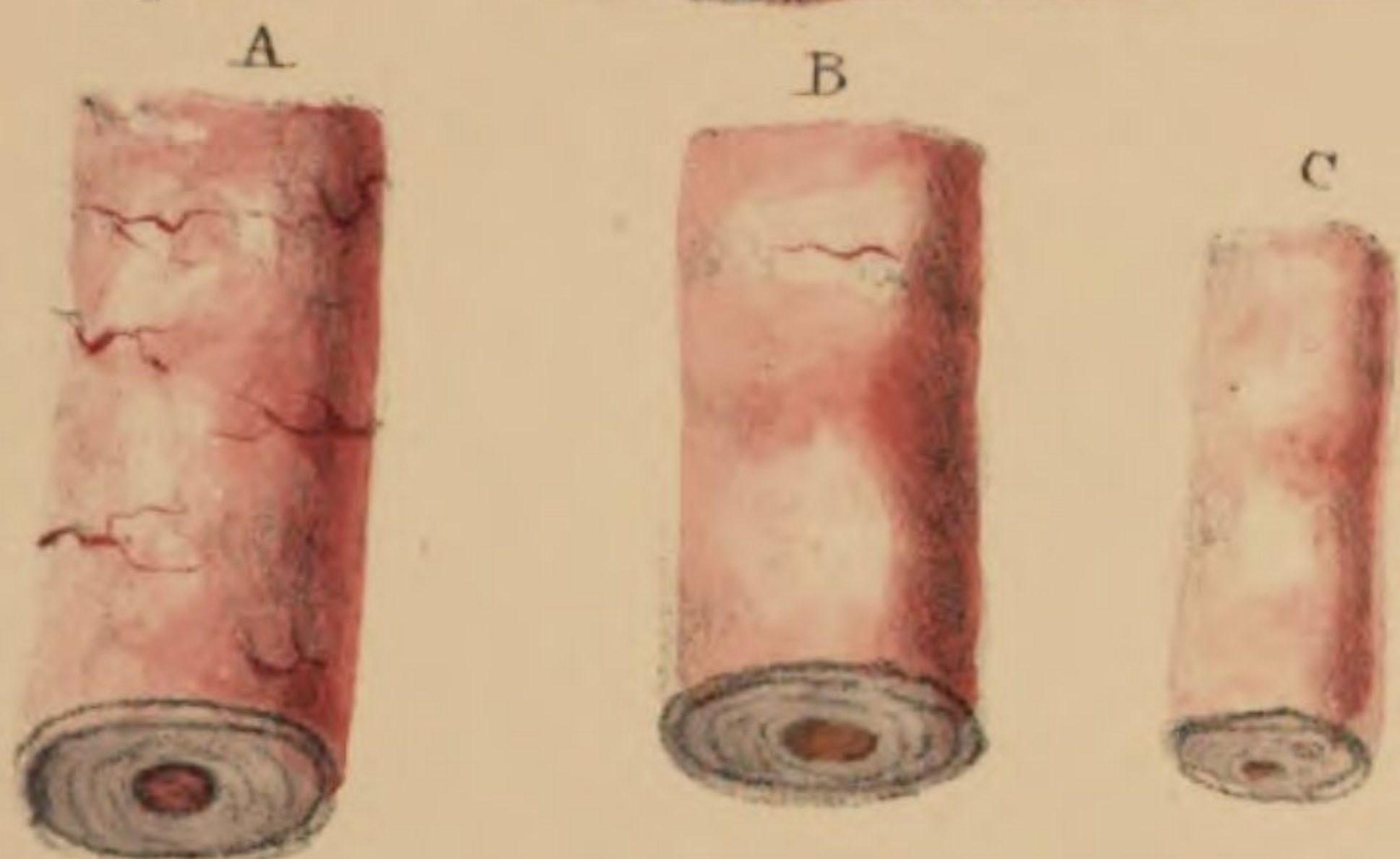


Fig. 8.

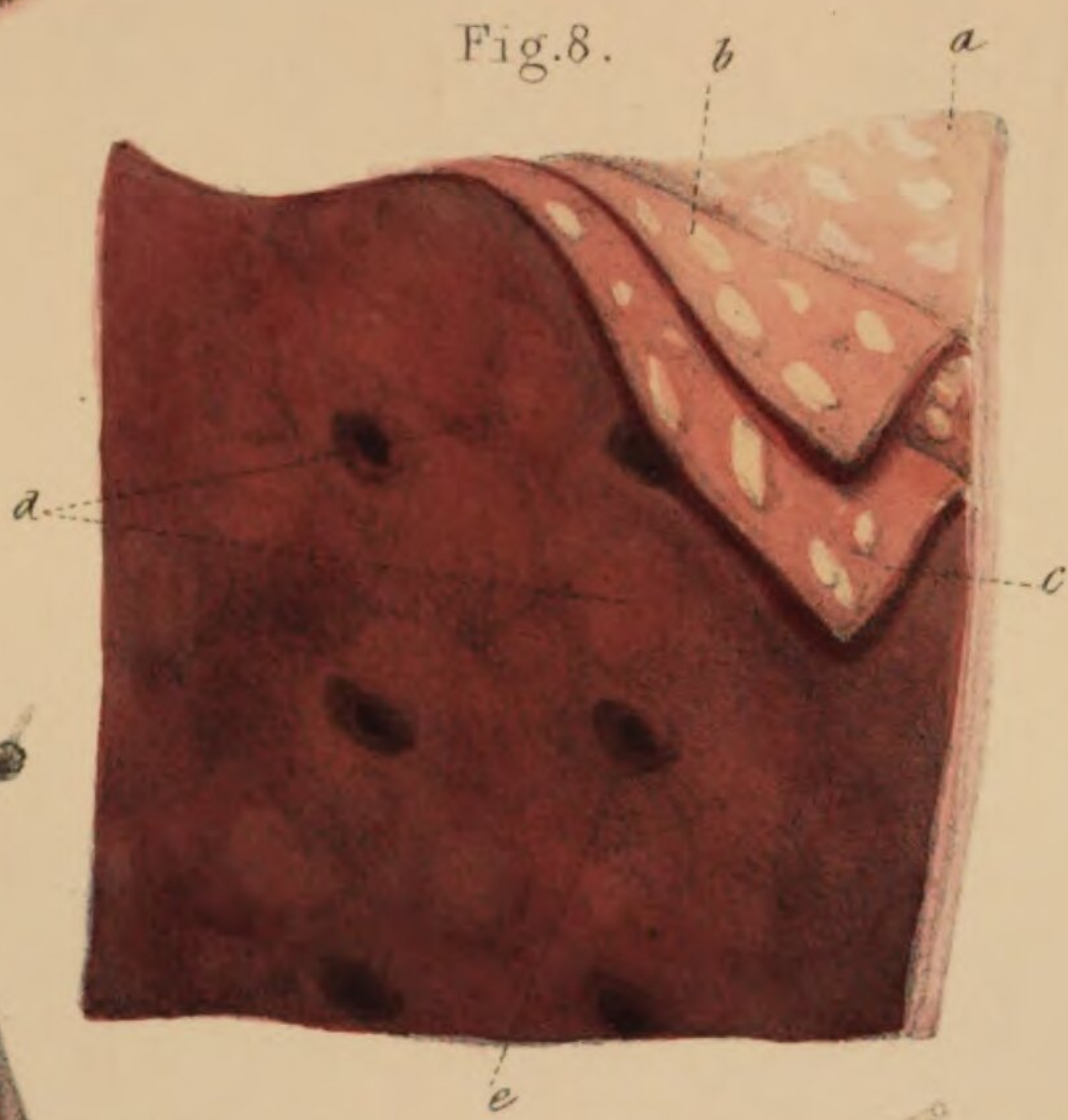


Fig. 3.

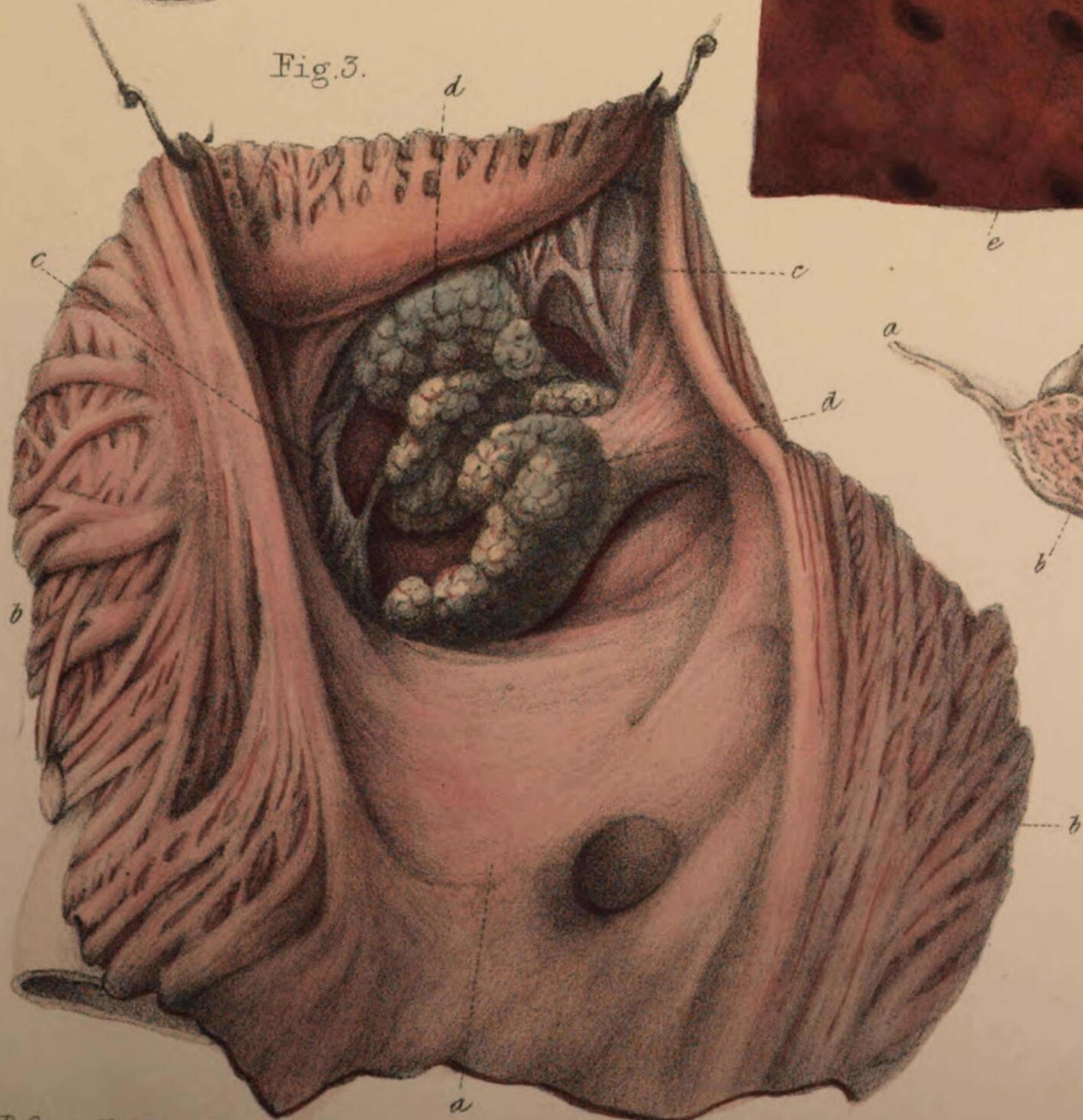


Fig. 5.



Fig. 4.



Fig. 6.

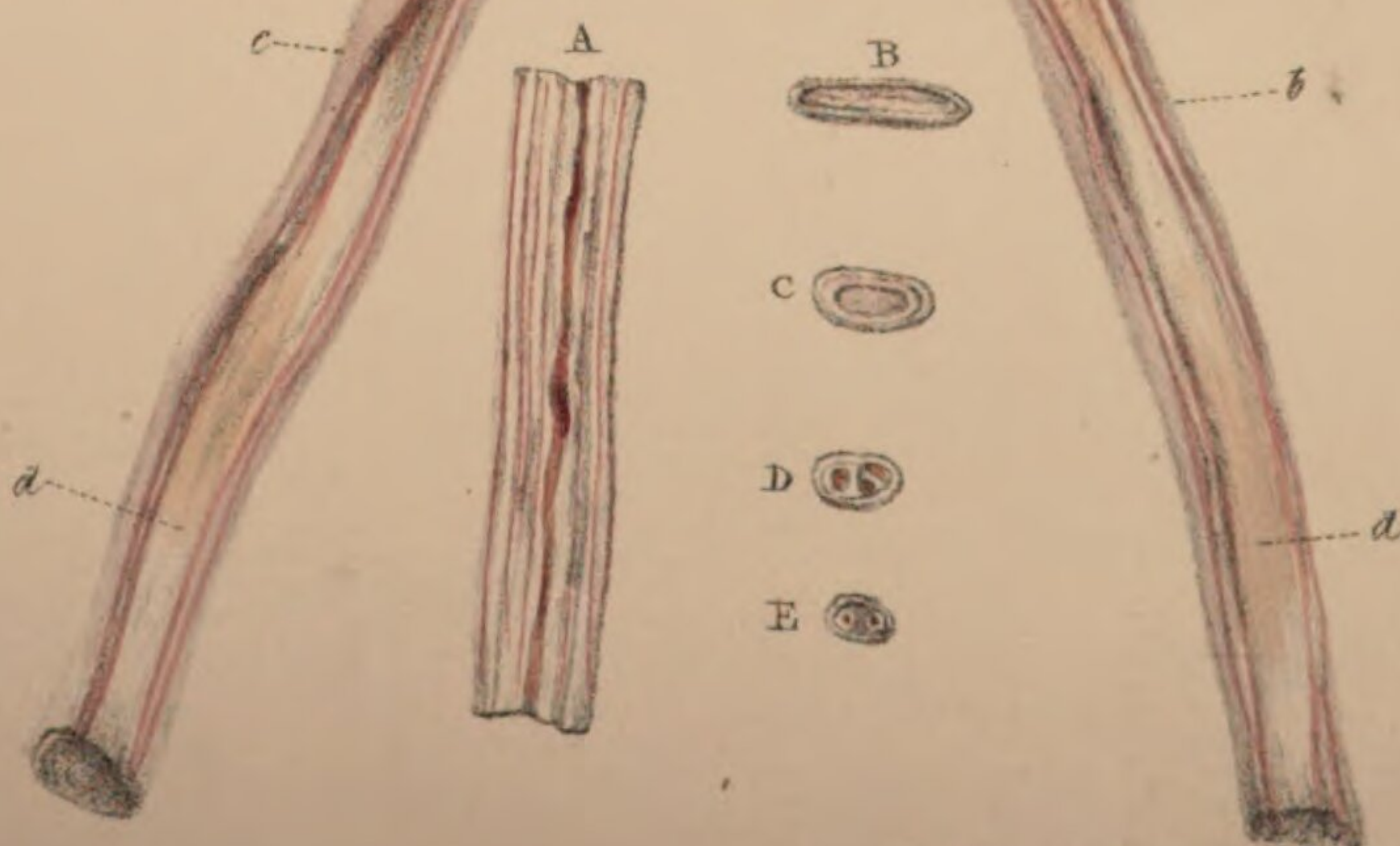


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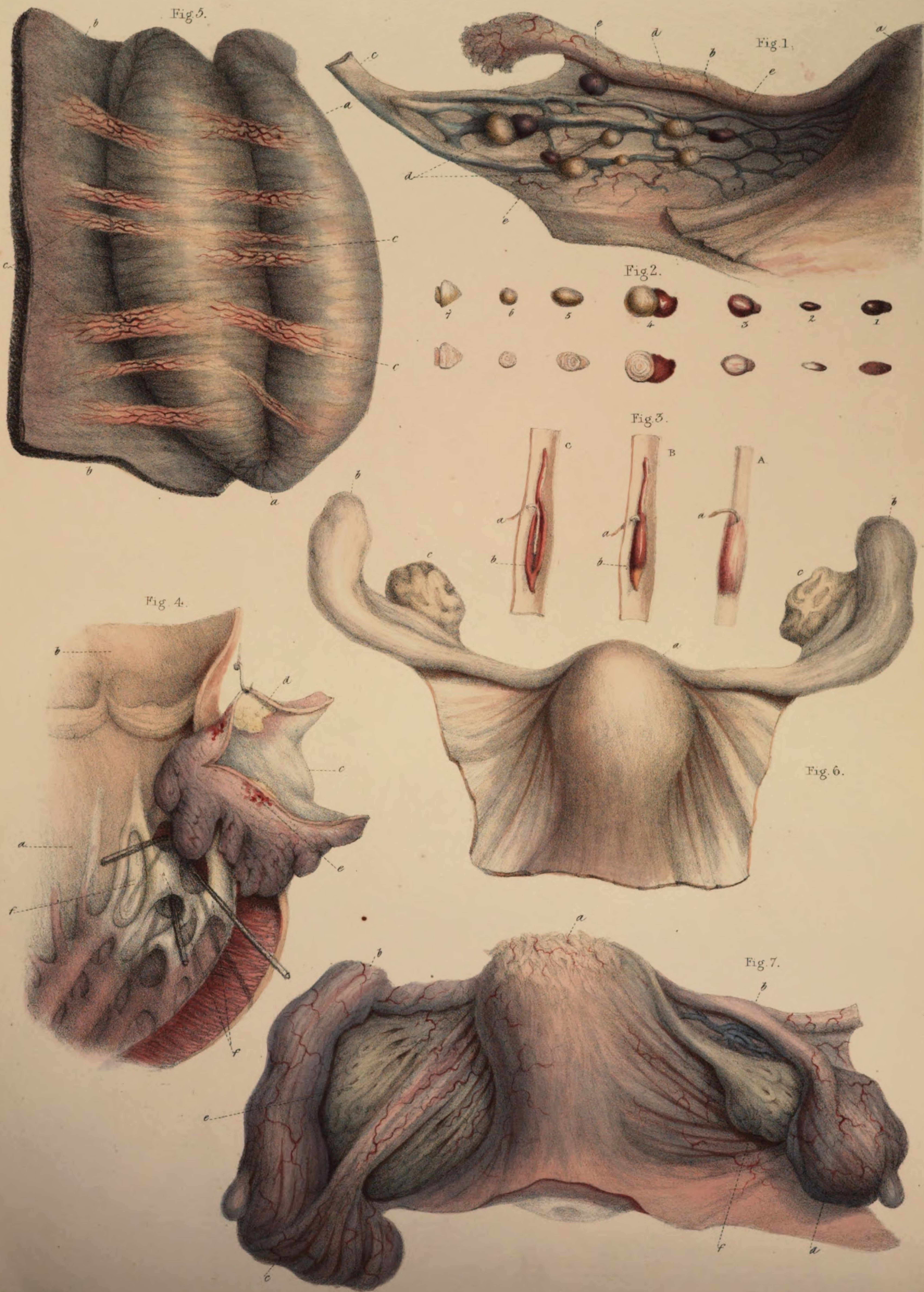


Plate IV.

Fig 7.

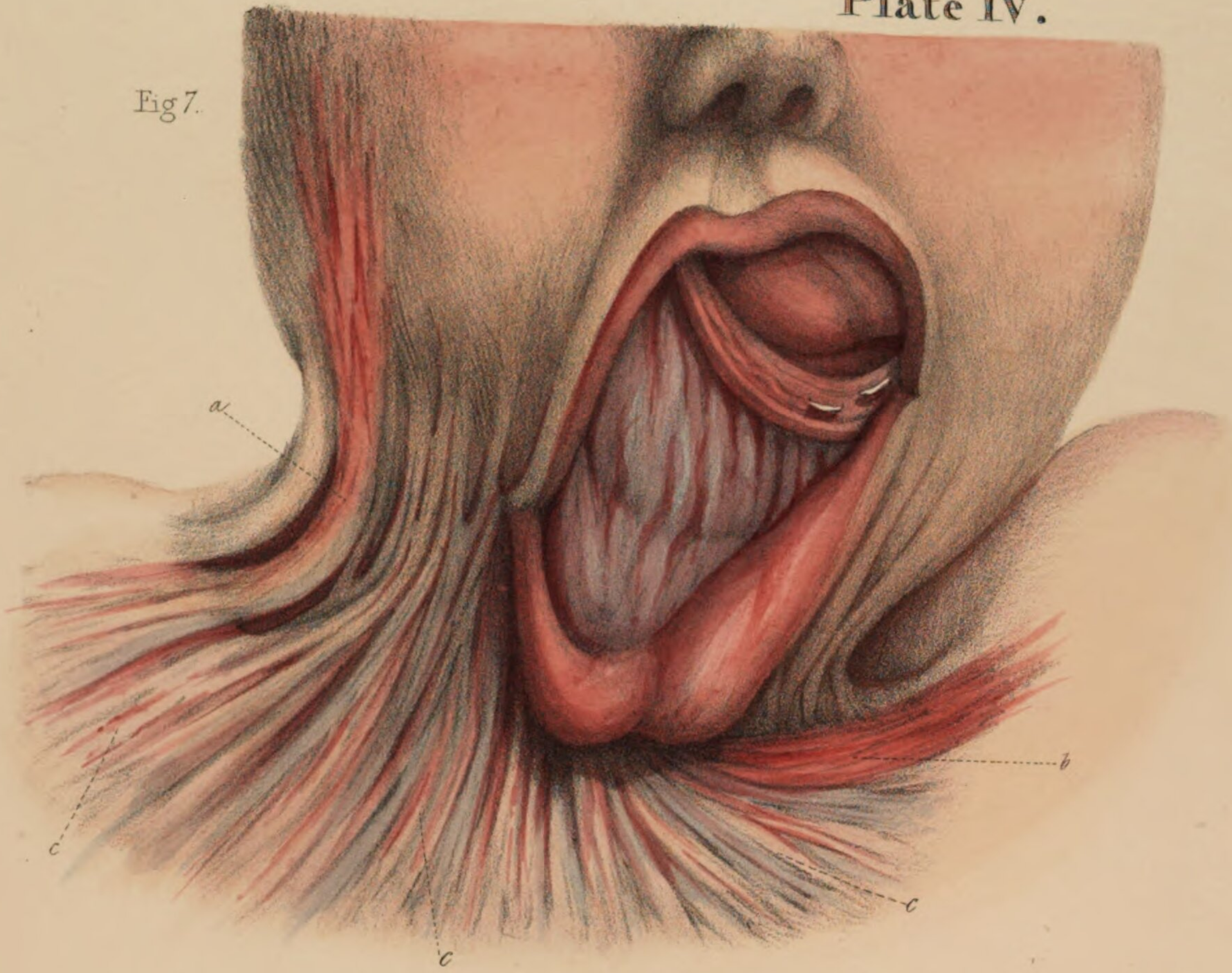


Fig 3.

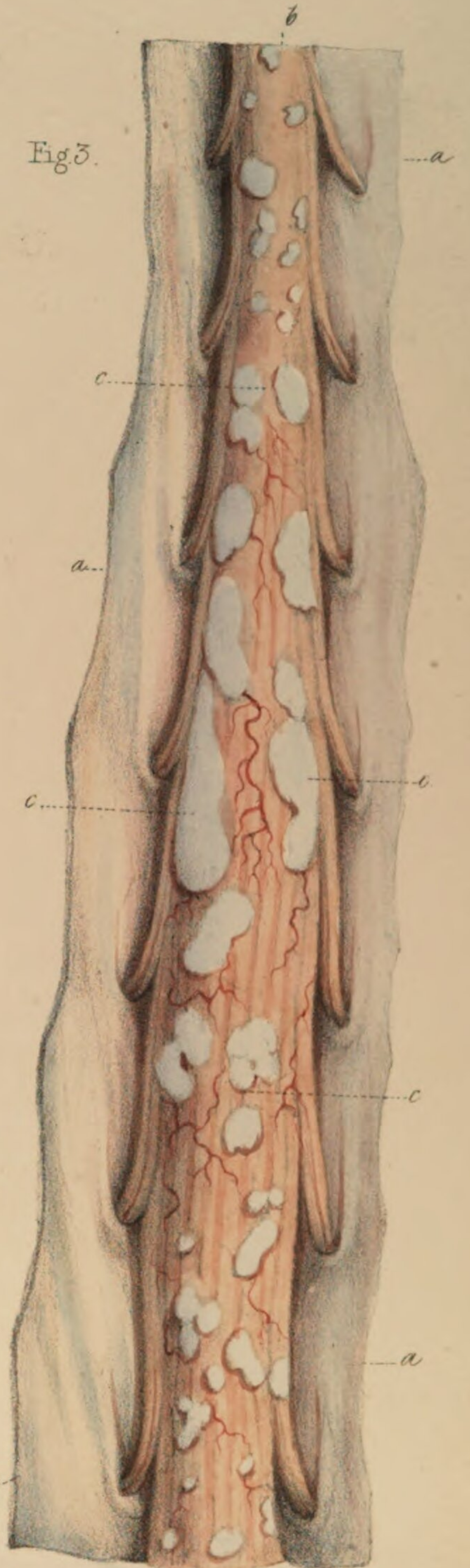


Fig 4.

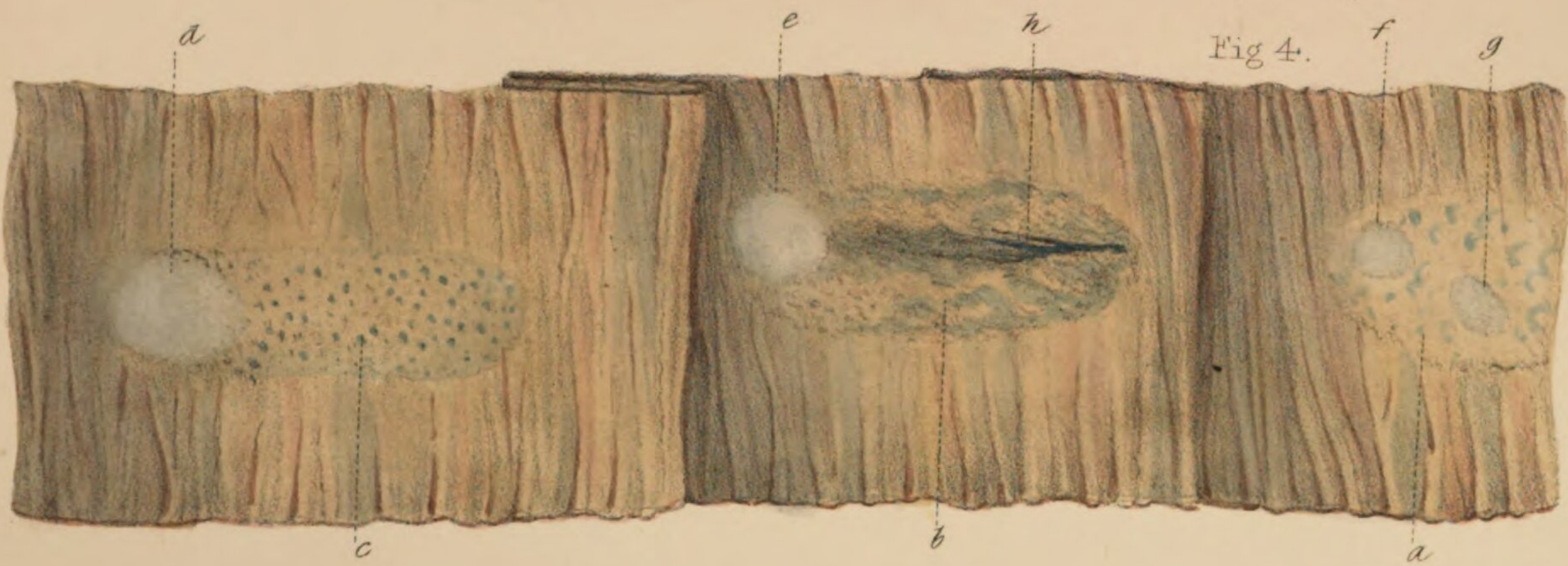


Fig 6.

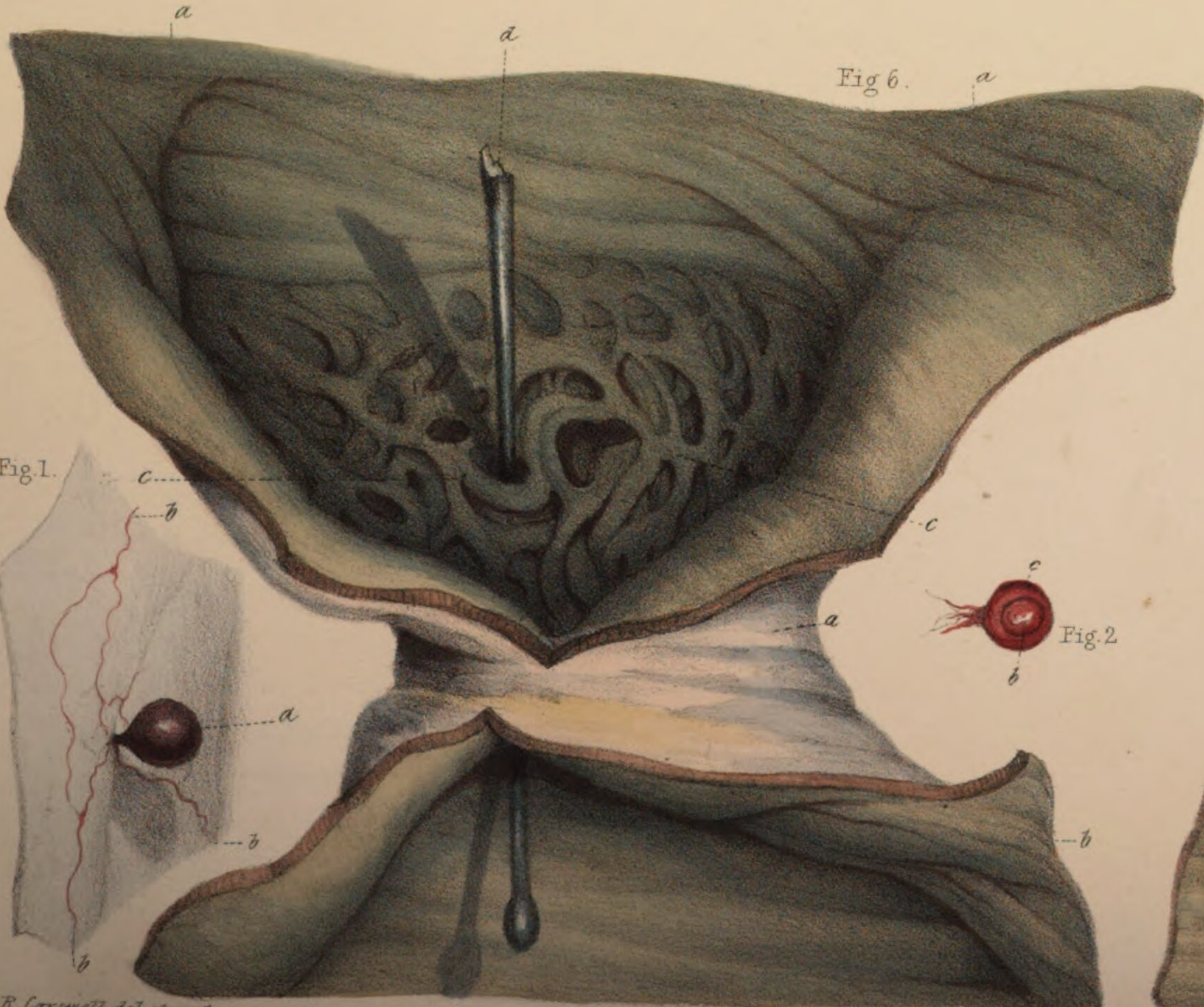


Fig 1.

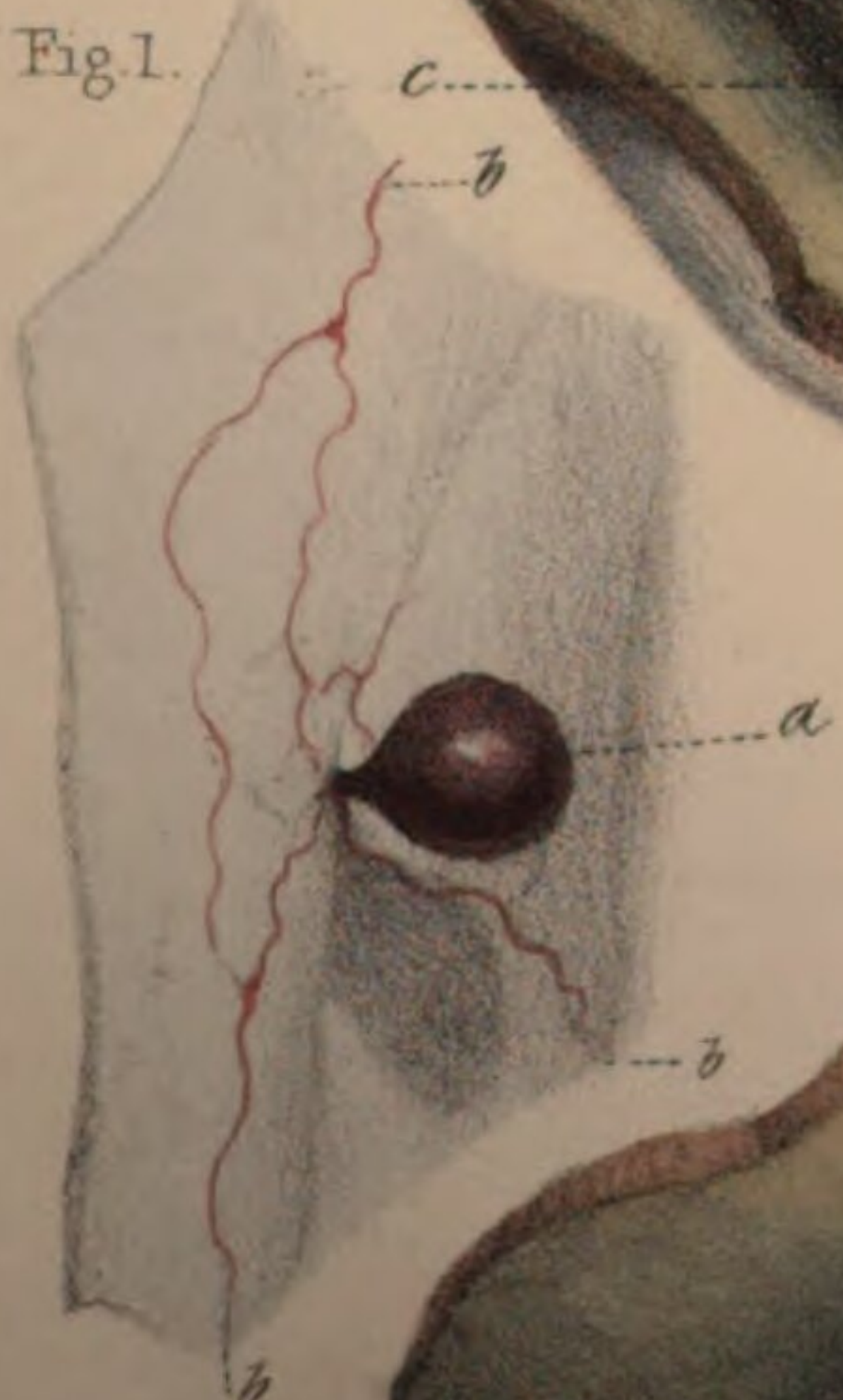
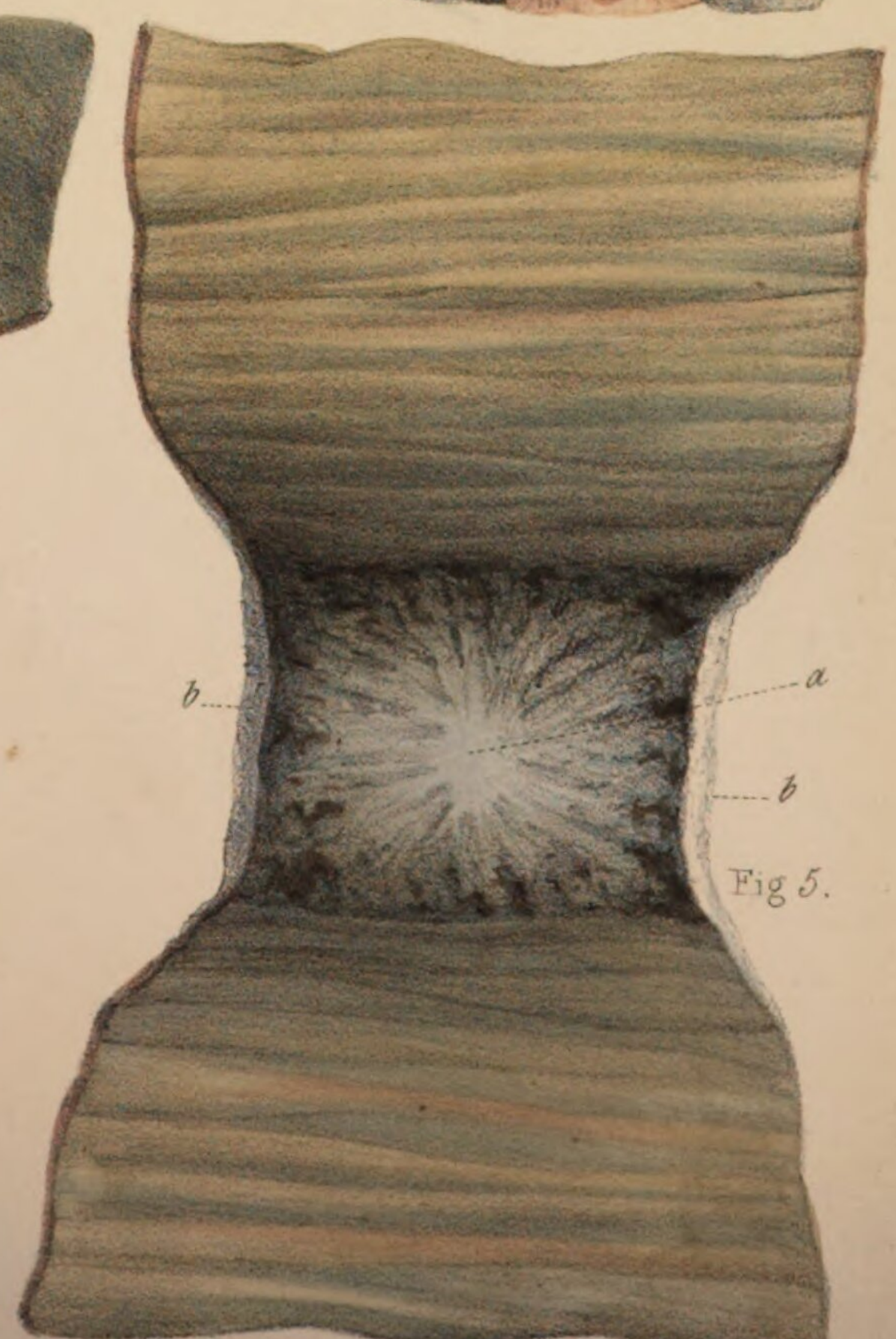


Fig 2.



Fig 5.



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ATROPHY.

It has already been stated in the preceding fasciculus that the modification of bulk which takes place in consequence of a diminution in the quantity of the solid materials which enter into the healthy composition of organs and tissues is termed Atrophy. The diminution of bulk which is thus produced, constitutes, therefore, the essential physical character of atrophy; and as the opposite modification of bulk, or hypertrophy, is referable to an increased exercise of the nutritive function, so must atrophy be regarded as depending on an opposite condition, or diminished exercise of the same function.

Nature, Origin, and Causes of Atrophy.—This condition of the nutritive function, and the subsequent diminution of bulk to which it gives rise, occurs under a variety of circumstances, the nature of which, as well as the influences which they respectively exercise in its production, are highly deserving of the consideration which they have received from recent pathologists. Various kinds of what are vulgarly called *monstrosities* are so many forms of congenital atrophy, occurring as the consequences of the formative process having been arrested at certain periods of the evolution of particular organs, which are thereby prevented not only from acquiring their normal bulk and capacity, but likewise present various modifications in their forms, relations, number, &c. all of which constitute subjects of deep interest to the physiologist, who has deduced from the study of them some of the most important laws which govern the development of the various classes of animals. A diminution of bulk, or the complete disparition, soon after birth, of organs which performed functions essential to the existence of the fœtus, but which are now no longer necessary to the maintenance of life, afford also interesting examples of atrophy. Thus the ductus arteriosus, umbilical arteries, and other blood-vessels peculiar to the fœtal circulation, become closed soon after birth, from the course of the blood through them being either interrupted or directed towards other organs, the exercise of which has commenced at this period. These vessels are afterwards converted into impervious cords, of which frequently no traces are to be found in the adult or at a later period of life. The functions of the thymus gland and suprarenal capsules ceasing after birth, these organs gradually diminish in bulk or entirely disappear. Differing from these two forms of atrophy,—the former of which is the consequence of defective or deficient development, the latter of the cessation of a function subservient to a new state of existence,—is that which supervenes during an advanced period of life, as the fulfilment of a general law of nature, so conspicuously manifested in the limited duration of all organized beings, which, having attained the maximum of their development, decline after a variable period of time, and tend gradually towards decay. In

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some organs, more especially those of a cellular structure, *senile atrophy*, as it may be called, is carried to a great extent, and produces changes the effects of which are often very conspicuous in the numerous and sometimes complicated derangements of function to which they give rise. It is well known that the solid bulk or weight of the lungs in old people suffers very great diminution in consequence of atrophy of the walls of the air-cells and minute bronchial tubes, a change which may take place in separate portions or throughout the greater part of both lungs, and which may be carried to such an extent as to efface almost entirely the vesicular structure of these organs. The tendency to this kind of atrophy appears to manifest itself soon after the adult period of life, by an increase in the dimensions of the air-cells, which have become considerably larger than they are in youth, and much more so than in childhood, the increase in their capacity being, however, accompanied by a corresponding diminution in the thickness of their walls. As the atrophy advances with the increase of age, portions, or the whole of the walls of a greater or less number of the continuous air-cells and corresponding bronchial tubes disappear, and give rise to the formation of a loose, irregular, cellular structure, in which no trace of the vesicular character of the lungs is perceptible. The cells thus formed vary from the size of hemp-seed to that of a pea or more, and are intercepted by imperfect septa, on which but few bloodvessels are conspicuous. The substance of the lungs is of a dark colour, from the languid circulation of the blood through them, and the imperfect decarbonization of this fluid, as a necessary consequence of these changes, and which also extend their influence to the form and dimensions of the thorax, both of which, as well as the functions of respiration and circulation, and the temperature of the body, are variously modified. The spongy structure of the penis has been described by Mons. Ribes as undergoing changes somewhat similar to those observed in the lungs. The spleen, however, although a cellulo-vascular organ, is not affected in this manner by the progress of age, but it is frequently much diminished in size, being sometimes not larger than a walnut; it is then hard, contains little or no blood, and is composed chiefly of cellulo-fibrous tissue and obliterated bloodvessels. Glandular organs, more especially the testes, lymphatic and mammary glands, are greatly reduced in size in some old people. The uterus is shrunk and very hard, and the ovaries are often transformed into a small mass of corrugated cellulo-fibrous tissue. The bones in general lose much of their weight, and become spongy and fragile; portions of some thick flat bones are reduced to a thin diaphanous plate; and fracture of the neck of the thigh-bone appears sometimes to have occurred in consequence of senile atrophy. The brain, spinal cord, and nerves, participate in the general decay of old age; the brain, after seventy, according to Desmoulins, being diminished from one-fifteenth to one-twentieth of its average weight, at the same time that it is specifically lighter than at forty years of age, and that the trunks and branches of the nerves are considerably less in size than in the adult. The longitudinal and transverse diameters of the brain in old age are stated by Cazauviehle to be several lines less than in the adult, and that the size of the corpora striata, optic thalami, and pons Varolii, is sensibly diminished, whilst that of the cerebellum remains unaltered even at the most advanced period of life. The muscles, especially those of voluntary motion, participate to a great extent in the general decay of old age, and the vascular system, if not the first in order to suffer from senile atrophy, is certainly always present in that of every other system or organ, and to which it bears a proportionate relation, either in the diminution which has taken place in the size of the large, or in the number of the small arteries.

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Of those forms of atrophy which occur after birth, some affect the whole body, others are confined to particular organs or tissues. The former, which require only a passing notice, are known under the appellations of marasmus, emaciation, &c. and follow as the remote consequences of various acute and chronic affections, particularly of the respiratory and digestive organs, or from the long-continued operation of causes which prevent the blood from undergoing those changes which render it fit for accomplishing the important function of nutrition, or by which its quantity merely is reduced, whether in consequence of defective nourishment, excessive evacuations, frequent hæmorrhage, and the like. The latter or local forms of atrophy, which, in general, are met with as permanent pathological conditions, may, in reference to the causes by which they are produced, be included under the three following heads :—1st, Atrophy from a diminished supply of blood ; 2d, Atrophy from the diminished exercise of the function of innervation : 3d, Atrophy from the diminished exercise of the functions of an organ.

Of the several forms of atrophy which have been thus generally noticed, I shall treat only of the latter, or those which are confined to particular organs or tissues, and which occur after birth as pathological states interfering with the regular performance of their functions. Those which originate in a modification of the formative process, or other causes operating before birth, will form a separate subject of consideration, although there are some of them, more especially those which affect the brain, and which are compatible with extra-uterine life, which would have been treated with advantage here.

1. *Atrophy from a diminished supply of blood.*—A diminution in the quantity of arterial blood necessary for the growth or perfect and vigorous accomplishment of the function of an organ, is one of the most obvious and immediate causes of atrophy. When the blood is prevented from being freely transmitted to a part of the body, by compression of, or adventitious products contained within, its principal arteries, it generally procures for itself a new passage, by means of collateral branches, through which it is carried in sufficient quantity for the perfect nutrition of that part. When this, however, is not accomplished by a collateral circulation, a greater or less degree of atrophy follows, as in the brain, in some cases of ossification of the carotid and vertebral arteries within the cranium ; in the spleen, the uterus, &c. from the same morbid condition of their respective arteries ; and not unfrequently in the inferior extremities, from this and other diseased states which interrupt the circulation of the blood through the larger arteries of these parts. Complete atrophy of the testicles follows obliteration, from disease, of the spermatic arteries, and from ligature of these vessels for the cure of varicocele ; and the removal of tumours, more especially those of the erectile kind, is sometimes obtained by cutting off the supply of blood to them, either by ligature of their principal arteries, or by repeated incisions, punctures, or the application of stimulating substances, the ultimate effect of which is obliteration of the arteries and veins of which these tumours are composed. The formation of accidental products contributes also to the production of atrophy in neighbouring tissues, by their withdrawing from these the quantity of blood necessary to their parasitical existence. Atrophy, however, from this cause alone, is never considerable.

Compression, produced in a variety of ways, of the capillary vessels is a frequent cause of this kind of atrophy. The stunted growth of some parts of the body, as the feet in some women, and the diminished dimensions of the inferior diameter of the cavity of the thorax in others, are partly produced by compression, so applied as to reduce the

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quantity of blood usually transmitted through the capillary vessels of these parts, although, in both cases, the comparative state of inactivity in which the muscles are placed by this cause, contributes greatly to the same effect. The liver, in some old women who employ a string or cord to fix their petticoats around the waist, presents, on its convex surface, a transverse fissure, which is sometimes from half an inch to an inch in depth; or the ribs, compressed by the same cause, leave deep impressions of their form on the surface of the same organ. The compression exercised by numerous solid and fluid accidental products, when slowly formed, gives rise to various degrees of atrophy of the surrounding tissues. Thus, tumours of the dura mater and bones of the cranium which project inwards not unfrequently produce cavities in the corresponding portions of the brain, equal in extent to the size of these tumours, which is sometimes considerable, thus prolonging the life of the patient to an indefinite period, although the atrophy alone, when carried to a certain extent, may give rise to partial paralysis. In like manner encysted tumours and abscesses, or serous cysts, formed in the substance of some organs, as in that of the liver, and in the cavities of others, as in the pelvis of the kidneys, are frequently accompanied by various degrees of atrophy. The greater part of one lobe of the former sometimes disappears in cases of chronic abscess, serous or suppurating cysts; and the substance of the latter has been found almost entirely removed, by the pressure of serous cysts, or that which is occasioned by the gradual accumulation, in the pelvis and infundibula, chiefly of serosity or pus. The formation of serous cysts in the kidneys is, however, by far the most frequent cause of atrophy of these organs, and as such cysts almost always occupy both kidneys, they give rise to a degree of atrophy which is not unfrequently followed by partial, and sometimes even total suppression of the secretion of urine. In one case of the latter description, which is represented in Plate I. *figs.* 4 and 5, both kidneys were crowded with serous cysts, varying from the size of hemp-seed to that of a small cherry; one of them preserved its natural dimensions, but a small quantity only of the cortical substance remained; the other was reduced to the size of a walnut, and surrounded by a mass of fat, having the form and original bulk of the organ. What is called cystic sarcoma of the mamma and testis is always accompanied by atrophy of the glandular structure of these organs.

The accumulation of serosity in the cavity of the arachnoid, and in the pia mater between the convolutions of the brain, which occurs in the insane and in old persons affected with general paralysis, is accompanied by various degrees of atrophy of that organ, but more especially of the grey substance of the convolutions. In this form of atrophy we find the entire brain separated from the inner surface of the cranium by a greater or less quantity of serosity, which is also accumulated in the pia mater between the convolutions. The upper, lateral, posterior, and anterior surfaces of the hemisphere are the parts in which the effusion is most abundant and most frequently observed. It may be confined to the sulci of a few only of the convolutions, or occupy the greater number of them on both hemispheres; or it may be slight or partial on one hemisphere, and extensive or general on the other. The presence of the effusion, its situation and extent, are readily recognized by the peculiar colour of the membranes beneath which it is situated, which present the appearance of irregular patches of a milk-white colour. In these parts also the membranes are opaque and thickened, and are, in general, united together in such a manner that the effused fluid is retained within them as within a sac. On detaching them from the surface of the brain, the fluid, therefore, is carried away with them at the same time, and in the situation which

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it occupied, are perceived irregular depressions or excavations, situated between, and extending to the bottom of, the convolutions, which are variously reduced in bulk, being more or less diminished in breadth and depth, sometimes much more in the former than in the latter direction of their dimensions. The surface of the excavations, which consists of the atrophied convolutions, presents a smooth glossy aspect, and is formed by the cortical substance, the colour of which is generally paler than natural, and most frequently corresponds with an increase of its consistence. Sometimes, however, its colour and consistence are not altered, or the former may be redder than natural, and the latter sometimes diminished, either generally or in circumscribed spots. The white substance of the convolutions presents a proportionate diminution of bulk, but little alteration of its colour or consistence.

This form of atrophy of the brain is met with in the great majority of cases of insanity which terminate in general paralysis. The existence of the former and the occurrence of the latter must be regarded in the relation of cause and effect, and in many cases which I have examined, the progressive development and degree of the one strikingly corresponded with the extent of the other.

It is still a matter of doubt whether the atrophy depends on a diseased state of the convolutions themselves, such as irritation or chronic inflammation, succeeded by an effusion of serosity to supply the loss of substance which has been supposed to be thus occasioned; or whether it is the consequence of compression, produced by the effusion and accumulation of this fluid between the convolutions, from a similar disease of the membranes. Having included it under the head of atrophy from a diminished supply of blood, I am disposed to refer it to the latter cause. I have already stated that the membranes covering the atrophied convolutions are always thickened and opaque, and are so united as to retain the effused fluid within them. Under these circumstances it is obvious that compression and atrophy of the contiguous convolutions must be the consequences. That the diseased state of the membranes arises in a modification of function of the convolutions is more than probable, but the change which the cortical and medullary substances of which they are composed undergo, is certainly not unfrequently manifested by the physical characters of atrophy alone; whilst, on the contrary, the membranes present the more usual and obvious effects of chronic inflammation, with effusion, its almost constant attendant. It is, however, necessary to observe, that the convolutions are not unfrequently found much diminished in size and deformed, in similar cases of paralysis. But this diminution in their bulk ought not to be included under the head of atrophy, as it does not differ from that which occurs in every organ which has suffered a solution of continuity, followed by a loss of substance. Here it is obviously owing to circumscribed softening or capillary hæmorrhage, particularly of the cortical substance.

Atrophy of the spinal chord is also sometimes observed in conjunction with and depending on the same causes as that of the convolutions. It is, however, likewise produced by atrophy of the convolutions in a different manner, afterwards to be noticed.

The accumulation of air in the vesicular structure of the lungs, known under the appellation of pulmonary emphysema, gives rise on the same principle to a particular form of atrophy of these organs. Thickening of the mucous membrane of the smaller bronchi in particular, the retention of inspissated mucus, compression and stricture of these tubes, violent and frequently repeated attacks of coughing, and efforts of every kind which prevent the free exit of the inspired air, are the causes of this form of

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atrophy. Acting as mechanical obstacles, they impede the transmission of the air through the bronchi both during inspiration and expiration; but the muscles of the former being much more powerful in their operation than those of the latter, introduce a greater quantity of air into the air-cells than can be expelled at the subsequent expiration, which therefore gradually accumulates in them, distends them mechanically and ruptures them, or by the compression which it exercises on their capillary circulation, destroys them by a slow process of atrophy. It is only, however, when the emphysema is great in degree that atrophy contributes to its production, or rather its further increase. This is best seen in those cases in which a portion of emphysematous lung projects considerably, as frequently happens when it occupies the summit or anterior border of this organ. When emphysema depends simply on dilatation of the air-cells, it may occupy only a portion or the whole of one or both lungs; but when rupture of the air-cells has taken place to a great extent, whether from distension or atrophy, it is generally confined to a limited number of lobules. In the former case the air-cells acquire the size of millet-seed, hemp-seed, or a small pea, the bulk of one or both lungs being at the same time proportionally increased; in the latter, several of them, or even a few contiguous lobes are thrown into one cavity, sometimes sufficiently large to contain a pigeon's egg, presenting a number of laminated and filamentous intersections, which divide it partially into irregular compartments. It is, therefore, as I have stated, only in this variety of pulmonary emphysema, which is a chronic affection, that atrophy is conspicuous. It is not observed in interlobular emphysema, which is an acute affection, as the air is absorbed before it has time to produce atrophy by compression. (See Fasciculus IX. Plate I. *figs.* 4, 5, 6, and 7, for illustrations of the principal forms of emphysema of the lungs.)

There is also one form of dilatation of the bronchi which presents a remarkable example of atrophy of these tubes. It sometimes occupies bronchi of considerable size, but much more frequently the small ramifications or terminations of these, which are dilated into the form of globular sacs, varying from the size of a pea to that of a walnut. Some of them consist of a uniform smooth cavity; others present several anfractuositities, which generally happens when a number of contiguous tubes communicate with each other in consequence of complete atrophy of a portion of their walls. In this case they might be confounded with emphysema, were it not for the nature of their contents, which consist always of a viscid, straw-coloured, muco-puriform secretion. The tenuity of their walls is such that Laennec has aptly compared them to the outer skin of an onion, and through which the colour and vesicular structure of the lung is seen as distinctly as beneath the transparent pleura. Their lining or mucous membrane is remarkably smooth, sometimes of a red, but more generally of its natural colour, and is much more tenacious than healthy mucous tissue. This form of dilatation is generally confined to a small number of bronchial tubes; but it sometimes affects the greater part of a lobe, generally the upper, the vesicular structure of which is compressed, deprived of air, and in that state of flaccidity which it presents in the case of chronic pleurisy with effusion. (See Fasc. IX. Plate I. *fig.* 3.)

The last of the causes requiring special notice, which give rise to atrophy in consequence of the obstacle which they occasion to the circulation of the blood in the capillaries, is that which follows the formation of a contractile fibrous tissue, on the surface of some organs and in the interior of others. The origin of this tissue (the nature of which will be found illustrated under the head of Analogous Formations) in

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inflammation, its fibrous structure, and the contractile property which it possesses, are best exemplified in chronic pleurisy and peritonitis. I allude here to its origin in inflammation of the serous membrane of these cavities, for it is not unfrequently met with in the liver, where it gives rise to inevitably fatal consequences, without our having any other than its mere presence as evidence of its having originated in the same pathological state in this organ as it obviously does on the surface of these membranes. In chronic pleurisy the lung is at first compressed by the effused fluids, and may, when these are removed by nature or art, regain its former dimensions. But it frequently happens at some subsequent period, that the coagulable lymph which the effusion contains becomes organized, and forms a strong fibrous membrane over the whole surface of the pleura pulmonalis, which retains the lung in the situation and reduced condition of bulk in which it was placed by the effused fluids, whether these be removed or not. But a further reduction of bulk is effected by this membrane alone, which, in proportion as it acquires the fibrous character, contracts in all directions, and not only compresses still more the affected lung, but ultimately reduces it to such a degree of atrophy that hardly any of its original structure remains. In this extreme state of atrophy of a lung from this cause, the greater number of the bronchi and bloodvessels are obliterated and greatly reduced in size, the pulmonary is replaced by cellular tissue, and the whole enclosed in a dense fibrous capsule, varying from an eighth to a quarter of an inch in thickness. That these effects are to be attributed to the gradual contraction of the accidental fibrous envelope of the lung is put beyond all doubt by the occurrence of atrophy of one lobe or a portion only of a lobe, in some cases of circumscribed pleurisy, followed by the formation of this tissue. The atrophy in these cases being circumscribed could not be the consequence of compression exercised by a fluid contained in the cavity of the pleura. And, besides, the atrophy is confined to that portion only of the lung which is covered by the fibrous tissue, which sometimes appears as if strangulated by a zone of this tissue surrounding the greater part of a lobe, or including an entire lobe in the form of a flattened sac, thus reducing the bulk of the affected part as much as in the case of compression of the whole lung from the same cause, a remarkable example of which is represented in Plate I. *fig.* 1.

In chronic peritonitis terminating in the formation of a fibrous membrane on the surface of the peritoneal covering of the viscera, we have also interesting examples of atrophy from compression, involving sometimes all the organs contained in the abdominal cavity. The intestines are often found much reduced in capacity, grouped together into a comparatively small mass, and firmly fixed down to the spine; the stomach and urinary bladder are flattened and compressed against the surfaces with which they are in contact; the gall-bladder is contracted and nearly empty; even the liver and kidneys in some cases appear to have undergone a diminution of bulk; and the spleen, in general, is very small. This organ is, besides, often subjected to atrophy from falls and blows or other causes which give rise to inflammation of its serous covering, and the subsequent formation of a thick fibrous capsule. This capsule has been found partly converted into cartilage and bone, and containing only a small condensed portion of spleen. Stricture of the œsophagus and intestines, succeeding to ulceration of their muscular coat, is attributable to the operation of the same cause. I have met with several fatal cases of stricture of the intestines produced by cicatrices formed of this tissue, and succeeding to ulceration, originally situated in the glands of Peyer, which had destroyed the muscular coat around the whole circumference of the tube in that portion of it

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occupied by these glands. The patients had left the hospital in a state of imperfect convalescence from typhoid fever, and returned some months, in one case more than twelve months after, with all the symptoms of ileus, the progressive development and increasing severity of which, as the constriction of the tube advanced, were strikingly illustrated by the history of the cases, and left no doubt as to the nature and cause of the disease. The obliteration of veins after phlebitis, and of arteries after ligature or other means which excite inflammation of their cellular sheath, followed by the copious effusion of coagulable lymph, is also partly to be attributed to the contractile property of the fibrous tissue which is formed around them under these circumstances.

The production of atrophy from the development of the contractile fibrous tissue in the interior of organs is no where so remarkable as in the liver, although it is occasionally observed in a slight degree in some other organs. The liver, when affected with atrophy from this cause, is sometimes reduced to a fourth of its normal dimensions; its consistence generally increases with the diminution of its bulk; it appears shrunk, and has an irregularly rounded form, particularly at its edges, and the whole of its external surface is raised into round flat projections, varying from the size of hempseed to that of a pea or even a small cherry. Examined more narrowly, the round flat projections are found to be composed of several smaller ones, and these, again, of the individual lobules of the liver; so that the larger projections are formed of aggregated groups of lobules, each separated the one from the other by cellulo-fibrous or fibrous tissue, the quantity of which varies considerably, and is always greatest between the largest groups of lobules. The situation of this tissue, its distribution, the manner in which it gives rise to the tuberiform arrangement of the lobules, and the diminution observed in the bulk of the liver, are important circumstances in the pathology of this affection, and which are most satisfactorily illustrated by a careful examination of the changes which have taken place in the structure of the organ. When this has been exposed by incision, the cut surface presents the same tuberiform arrangement seen on the external surface beneath the peritoneal covering, the lobules being grouped into smaller or larger masses, mostly of a round, ovoid, or pyriform shape. The cellulo-fibrous or fibrous tissue now forms a conspicuous feature in the disease, both on account of its greater quantity compared with that of the lobular structure of the liver, and the contrast of its white or grey colour with the rust, yellowish or greenish brown colour of the lobules. It is seen occupying the sheath of the portal veins, following the whole course of these vessels, both in their passage to and their distribution between the lobules in which they terminate. It thus forms around the veins, in the former situation, a firm fibrous sheath, and in the latter a capsule enclosing a variable number of lobules, in some parts only four or six, in others ten, twenty, or more. Hence the obvious reason why the lobules are grouped together in the form of tumours of different sizes, containing or subdivided into smaller ones. In separating one of these groups of lobules or tumours from the surrounding ones, which can often be done with great facility, especially at the commencement of the disease, we find that it is held, at a certain point of its circumference, by the bloodvessels which pass into the lobules contained within it. At this point the vessels are obviously constricted by the fibrous sheath which surrounds them, and the lobules themselves by the same tissue which forms their common capsular covering. The interior of each group of lobules, when exposed by section, presents a number of fibrous intersections, continuous with the common capsule, and are obviously formed by this tissue where it surrounds the terminal divisions of the portal veins. The

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quantity of the fibrous tissue compared with that of the lobular structure of the liver in this disease varies greatly. At the commencement it is small in quantity, and is best seen where it surrounds the veins before they give off their terminal branches, and consequently where it forms the capsular covering described. In the progress of the disease it becomes more and more abundant, and at the termination of some cases forms the greater part of the bulk of the liver. In the same proportion also as it increases in quantity, does the lobular structure of the liver disappear and its bulk diminish, and so much is this sometimes the case, that almost no trace of the natural structure of the organ is observable. (See Plate II. *figs.* 1, 2, 3, 4, and 5.)

From what has been said of the situation and disposition of the fibrous tissue, in reference to the portal veins and lobules, the constriction of the former and the atrophy of the latter which are observed to take place in this disease will readily be understood. For here, as in the other organs to which I have alluded, this tissue must, from its locality, and in virtue of its contractile property, tend continually to diminish the capacity of the vascular structure of the liver, and consequently its entire bulk. The mechanical obstruction to which it gives rise is at first confined to the capillary circulation; but when the lobules in the progress of the disease are grouped together in the form of tumours, a new obstacle is created, which acts on the venous circulation of the liver in general, but more especially on that of the portal veins. For these tumours either compress the portal veins, or, projecting in the direction of their interior, render them so unequal and at the same time so narrow, that the circulation of the blood through them is always more or less impeded, and sometimes almost entirely interrupted. These effects of the tuberiform condition of the lobules are always much greater in the portal than in the hepatic veins, for this reason, that the attachment of the former to the lobular structure of the liver is very loose, on account of their being provided with a cellular sheath, which the latter have not. They also, however, undergo the same changes in a less degree, particularly the inequality of their internal surfaces from protrusion of the tumours. It is, perhaps, not unworthy of remark, that the tuberiform aspect of the surface of the liver is to be ascribed entirely to the contractile property of the fibrous tissue, for it has been supposed that it was produced by the development of a new tissue of a peculiar kind, and which was considered by Laennec to constitute this disease, to which he gave the name of *Cirrhose*, on account of the rust-brown colour which it so frequently presents. That it is not a disease depending on the formation of a new tissue, but on the contrary consists in quite an opposite state, viz. a state of atrophy, must be evident from the description which I have given of it. The only new tissue found in the liver is the contractile fibrous tissue, and as I have already said, in proportion as this increases, the lobular or proper structure of the organ diminishes. The reason, therefore, why the surface of the liver presents a tuberiform appearance is, that the fibrous tissue being attached to the peritoneal covering pulls this membrane inwards all around the groups of lobules, where, I have said, it is most abundant. The central portion of the groups of lobules must therefore, from this cause alone, become prominent, and must also be subject to further increase, from the accumulation of blood and bile in their vascular structure.

With regard to the state of the gall-ducts and hepatic artery in this disease, it is certain that as they pass to and from the lobules respectively, enclosed in the capsule of Glisson, they must also, particularly the former, like their accompanying veins, undergo compression. The secretion of the bile, however, is effected, although apparently

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reduced in quantity, without presenting any remarkable alteration of its physical properties.

The different colours which the liver presents when affected with atrophy of this kind are derived from the blood and bile accumulated in the lobules, the relative proportions of which varying in the progress of the disease, give rise to various shades of yellow, red, green, and brown; as orange-yellow; rust, reddish, and greenish-brown colours, as one or the other of these fluids predominate. The rust-brown and orange-yellow colours are most common at the commencement, the greenish-brown towards the termination of the disease.

In some cases of cirrhosis, the obstruction to the circulation of the blood through the portal veins gives rise to appearances very different from those which I have described as characterising this affection. As these vessels are compressed and narrowed in the manner I have stated, they are generally found to contain only a small quantity of blood after death. It occasionally happens, however, that they are filled or distended with a mixture of coagulated blood, fibrine, and bile, the presence of the two former in particular indicating that the circulation must have almost entirely ceased before death. In three cases of this kind which I have met with, the whole of the portal system, from the commencement of its trunk to its termination in the lobules, was in this state. A great many of the portal veins, both large and small, were enormously dilated, and some of the smaller ones appeared to be ruptured, so that, when cut across, they presented the appearance of bloody tumours or fungus hæmatodes, scattered throughout the substance of the liver. (See Plate III. *fig.* 1.)

In concluding the description of the physical characters of cirrhosis, I may remark that the nature of this affection affords a satisfactory explanation of the occurrence of ascites, which, to a greater or less extent, is its constant attendant. The extent of the serous effusion varies with the degree of compression to which the portal vessels are subjected by the fibrous tissue. Thus, at the commencement of the disease, when the quantity of the fibrous tissue is small, the effusion is generally inconsiderable; whereas, at a more advanced period, or towards its termination, when this tissue has increased in quantity, and by its contraction opposed a still greater obstacle to the return of the blood from the chylopoietic viscera, it is often very great. Hence also, for these and other reasons already stated, the tuberiform character of cirrhosis may be well marked at the commencement, although the effusion may be small in quantity, and may be much less apparent at the termination of the disease, in consequence of the atrophy of the lobular structure of the liver, which has taken place at this period, when the effusion is always greatest.

2. *Atrophy from the diminished exercise of the function of innervation.*—Whatever may be the manner in which the function of innervation is associated with that of nutrition, it is a well-known fact that various diseases and injuries of the brain, spinal chord, and nerves, which interrupt or suspend for a considerable length of time the exercise of this function, are always followed by a certain degree of atrophy of particular parts of the body. However, as paralysis always accompanies atrophy in these cases, we cannot, when the brain is the seat of the primary lesion, form a correct estimate of how much of the atrophy is due, respectively, to the diminished exercise of the nervous influence, or to the state of comparative inactivity of the affected part. In hemiplegia from disease of the brain, the paralysed limb or limbs, or even the body on the same side, are generally more or less wasted; and that the diminution of bulk

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which these parts suffer, is not the mere consequence of diminished muscular action, is proved by the fact that the one is not always in proportion to the other. This fact, and consequently the influence of suspended or diminished innervation in the production of atrophy, independently of that of volition, in voluntary muscles, is still more satisfactorily shewn by the occurrence of atrophy of a limb, from compression and other injuries which destroy or interrupt the function of its principal nerve, to a much greater extent and after a much shorter lapse of time than happens in paralysis from disease of the brain. Atrophy of the upper extremity from compression of the brachial plexus or nerves, in luxation of the head of the humerus, is an example of this kind. A remarkable case of atrophy of the right inferior extremity, from injury of the crural and sciatic nerves on that side, is recorded by Lobstein, which he observed in a man fifty-four years of age, who was thrown down in the street when a child. The right limb became soon after feeble, and gradually diminished in bulk as he advanced in years. The muscles, when examined after death, were pale, and reduced to the form of a fleshy membrane; the gastrocnemius and soleus muscles weighed only two ounces and six drachms, whilst those of the healthy limb weighed nearly eight ounces; the bones of the right side of the pelvis were considerably reduced in size and thickness; the right femur weighed only three ounces two drachms and a half, whilst that of the opposite side weighed nearly five ounces seven drachms.

Similar forms of atrophy, accompanying paralysis, occur sometimes in portions, or particular muscles only, of a limb, which show that the cause must be confined in its operation to the nerves which are in communication with the paralysed and atrophied muscles. Of this kind is the atrophy which accompanies painters' colic, and some of the worst forms of what is called *dyspepsia*, in highly nervous, hysterical, and hypochondriacal individuals. There can be no doubt that in such cases the atrophy is, at least in part, the consequence of the same morbid condition of the nerves which gives rise to the paralysis, and which, acting on the capillary vessels, either retards the circulation of the blood through them, or prevents this fluid from undergoing the changes necessary to the accomplishment of the regular process of nutrition.

3. *Atrophy from the diminished exercise of the functions of an organ.*—Atrophy from the diminished exercise or the total suspension of the functions of an organ, occurs under a variety of circumstances. Lesions of the brain, spinal chord, and nerves, followed by paralysis, give rise to this form of atrophy, in consequence, as has already been observed, of the diminished power of motion in the affected muscles. Although it is chiefly in the muscles of voluntary motion that it is observed, it is also met with in those of involuntary motion, particularly in the intestines and bronchi. In the former, it is sometimes carried to a great extent in that portion of the intestine through which, in *artificial anus*, the food or fæces have long ceased to pass; the intestine being contracted upon itself, greatly diminished in bulk, and its muscular coat reduced to a thin transparent membrane. In the latter, it is still more frequent in its occurrence, from a variety of diseases to which they are liable, and to which I shall presently allude. It is asserted that the muscular substance of the heart, in hypertrophy of this organ, undergoes a considerable diminution of bulk, under the treatment of Valsalva; and Laennec relates a case of this kind in a woman, fifty years of age, in whom, having died of cholera two years after recovery from the disease, the heart was found of the usual size of that of a child twelve years old, and presented, externally, the appearance of a withered apple, the wrinkles running longitudinally.

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Causes which permanently interrupt the passage of the air through the bronchi, as tumours which compress these tubes, accidental products contained within them, cicatrisation and constriction of their walls, or which prevent for a considerable length of time the free and full expansion of the lungs, as incidental fluid and solid products contained within the cavity of the pleura, vicious modes of dress, as well as all those occupations and habits which limit the action of the respiratory muscles, or which retain these organs in a state of comparative inactivity, give rise to partial or general diminution of bulk of one or both lungs. When a portion of a bronchial tube becomes obliterated by any of the causes which have been enumerated, the remaining portion of it, or the terminal branches of which it is composed, frequently undergo a gradual diminution of bulk, become likewise obliterated, and are ultimately converted into solid fibrous cords. Sometimes only one bronchus, but more frequently several bronchi, either in one or both lungs, are found obliterated in this manner, and the portions of pulmonary tissue to which they are distributed entirely deprived of air, collapsed, flaccid, and atrophied. An interesting memoir has lately been published by my friend Mons. Reynaud, illustrating several of the forms of this kind of atrophy, or rather *obliteration* of the bronchi, which he has shewn to be much more frequent in its occurrence than had been supposed by other pathologists. The bronchial tubes thus effected are seldom larger than those of the third or fourth order, and, therefore, the atrophy which takes place in consequence of their obliteration is seldom accompanied by a marked diminution in the bulk of the lungs, unless several of them are simultaneously affected. When this happens, however, an obvious diminution of bulk of the affected lung is observable, the external surface of which presents an irregular, lobulated appearance, in consequence of the healthy portions of the pulmonary tissue into which the air is freely admitted, being raised above those from which it is excluded, or which are in a state of atrophy, from the impervious condition of their bronchi.

It is not rare to meet with compression of larger bronchial tubes than those alluded to, by enlarged tuberculated glands, in children; but, although such causes of compression sometimes impede the function of respiration to a great extent, I am not aware that they have been observed to give rise to atrophy of the pulmonary tissue. The only analogous case of this kind which I have seen, occurred in a monkey which I had an opportunity of examining along with Mons. Reynaud. The left division of the trachea was surrounded by a mass of tuberculated bronchial glands as large as a walnut, in the centre of which it was embedded, and so compressed as to be rendered completely impervious to the passage of the air. The corresponding lung was reduced to nearly one-fourth of the size of the other, and as the cavity of the pleura contained no fluid, the parietes of the chest on this side were depressed in the same manner, as in cases of chronic pleurisy with permanent compression of the lung, after absorption of the effused fluid. Both lungs were crowded with groupes of tubercles of various sizes, and the right, that by means of which the function of respiration had been, for some considerable time, exclusively performed, was extensively emphysematous. (See Plate IV. *fig. 3.*)

A considerable number of cases of atrophy of the gall-bladder, arising in a diminished exercise or the total suspension of its function, have been recorded. Those which I have seen depended either on obliteration of the cystic duct from enlarged lymphatic glands, or a preternatural communication existing between the gall-bladder and a neighbouring portion of intestine, through which gall-stones had made their

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escape. In the first case, the bile is prevented from arriving at the gall-bladder; in the second, instead of accumulating in this organ, it passes gradually into the intestine. Hence, being no longer distended by the bile, the gall-bladder contracts upon itself, becomes shrunk, and, in some cases, reduced to a small nodule of cellulo-fibrous tissue.

The remaining organs requiring notice, in which atrophy occurs in consequence of the suspension or cessation of their special functions, are those of generation, especially the mammæ and testes, the brain, spinal chord, and nerves. It is well known that the mammæ seldom acquire the same magnitude in females who have not suckled as in those who have; and the great diminution which they undergo, when the exercise of their function is no longer required, affords a striking illustration of the influence of the suspension of a physiological act, in the production of atrophy. The testes, also, furnish us with examples of atrophy equally remarkable, although not always equally well understood. In persons who have spent their youth under the humiliating and blighting influence of certain monastic vows, the testes either remain in a state of imperfect development or become wasted; and Baron Larrey asserts that injuries of the cerebellum, from wounds occupying the region of the occiput, are also followed, at some period after recovery, by atrophy of the testes. Although I am not aware that similar cases have been observed by other authors, it does not appear to me that there are any grounds for disbelieving the Baron's statement, more especially as it has been ascertained that the special function of these organs, if not dependent for its accomplishment on the physiological exercise of that of the cerebellum, is certainly either increased to a remarkable degree, or altogether controlled by various morbid conditions of this organ. Many examples have been recorded which prove that such a functional relationship exists between the testes and cerebellum; and I may add, in further confirmation of the fact, that I have met with two cases in particular, in which this relationship was manifested in a most remarkable manner. They occurred in two young men, from eighteen to twenty years of age, who reduced themselves to a state of the most appalling moral and physical degradation by the act of self-pollution. Both of them died from its effects; one of them having often declared that he was compelled towards the gratification of a desire which he had no power to control, for he had frequently attempted the consummation of it after the prepuce had been excised as a means of prevention, and when the glans and part of the penis were in a state of active inflammation. In each of these patients the cerebellum was the seat of a tumour as large as a hen's egg, composed entirely of the medullary sarcoma.

There are no facts which prove that the brain, or particular portions of it, undergo a diminution of bulk, merely in consequence of diminished activity of their functions. It is only in the case of disease affecting the convolutions of the brain, that we meet with conspicuous examples of atrophy of some of the central portions of this organ. In some of the cases of atrophy of the convolutions which I have described as occurring in general paralysis of the insane, the corpus striatum and optic thalamus were much diminished in size, either on one side or both, according as the former occupied one or both hemispheres: nor was the secondary occurrence of atrophy confined to these portions of the brain; it extended to the crura cerebri, pons Varolii, medulla oblongata, and spinal chord. In one of these cases, the atrophy of the convolutions was much more extensive on the left than on the right hemisphere, and the corpus striatum and thalamus had also suffered a greater diminution of bulk on this than on the other side.

In a case of apoplexy which had destroyed nearly the whole of the left corpus
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striatum, I found the corresponding crus cerebri, one half of the pons Varolii and medulla oblongata, together with the corpus pyramidalium and olivarium, reduced to nearly one half the size of the same parts on the opposite side.

I have met with two cases of a remarkable lesion of the spinal cord accompanied with atrophy. One of the patients was under the care of Mons. Louis, in the Hospital of La Pitié, the other under the care of Mons. Chomel, in the Hospital of La Charité, both of them affected with paralysis. I did not see either of the patients, but I could not ascertain that there was any thing in the character of the paralysis or the history of the cases, calculated to throw any light on the nature of the lesion found in the spinal cord. I have represented the appearances observed in one of these cases in Plate IV. *fig. 4*, in which the pons Varolii was also affected, and which convey an accurate idea of the physical characters of the lesion. In this case a distinct portion of the cord was affected with softening, which of itself would no doubt have accounted for the paralysis; but in the other case there was no other lesion present than that to which I allude, to which the paralysis could be attributed. The anterior surface of the spinal cord presented a number of spots, from a quarter of an inch to half an inch in breadth, of an irregular form, of a yellowish brown colour, smooth, glossy, without vascularity or any alteration in the colour or consistence of the surrounding medullary substance. The medullary substance thus affected was very firm, somewhat transparent, and atrophied. At the root of the medulla oblongata, these changes occupied the whole breadth of both the medullary fasciculi to the extent of half an inch in breadth from above downwards. Further down, they were confined to distinct spots on each fasciculus, and several of the same kind, but smaller, occupied the pons Varolii. The depth to which the medullary substance was affected in this manner varied from half a line to three or four lines, and on dividing the cord, it was seen to penetrate as far as the grey substance.

It is a singular fact that the nerves of muscular organs which have long been affected with paralysis, seldom present any very sensible diminution of their bulk. It is, perhaps, only in the optic nerves that atrophy has been observed as the consequence of disease or injuries of one or both eyes, which either render vision impossible or extremely feeble for a great length of time. The extent of the atrophy, under these circumstances, varies considerably, as the lesion of the function of vision has been more or less complete, of shorter or longer duration. The atrophy, in the great majority of cases, is confined to that portion of the nerve situated between the eye and the optic commissure; and in those cases in which it extends beyond the latter point, the most generally received opinion is, that it is the nerve on the opposite side that is affected. When the atrophied nerve is examined, it is found to be variously reduced in bulk; the nervous matter in particular is very small in quantity, or has altogether disappeared. The cellular and fibrous tissues of the nerve, consequently, become more apparent, constitute, in some cases, the greater part of the nerve, or occupy, in others, its place, in the form of a fibro-cartilaginous cord.

DESCRIPTION OF THE PLATES.

PLATE I.

Figs. 1, 2, and 3, represent atrophy of the lung from the compression to which it is submitted by the presence of an effusion into the cavity of the pleura, or the formation of a fibrous membrane on the pleura pulmonalis. In fig. 1 is represented the partial or circumscribed atrophy, which I have described as sometimes taking place, when the inflammation of the pleura, the effusion of the coagulable lymph, and its subsequent organization and conversion into a fibrous membrane, are confined to a single lobe, or a portion only of a lobe. A, A, inferior half of the upper lobe of the right lung; B, B, the inferior lobe, entirely inclosed in a strong covering of fibrous tissue, and greatly reduced in bulk; C, C, the lower portion of the upper lobe surrounded by a tissue of the same kind, and compressed into the form of a cylindrical projection about the size of the little finger, at the extremity of which is seen a small healthy portion, E, of the anterior extremity of the lobe, uncovered by, and projecting beyond this tissue at the point D, where its constricting force is most apparent. Figs. 2 and 3 represent a peculiar change produced in the pleura pulmonalis, in acute inflammation of this membrane, followed by effusion and compression of the lung. The pleura cannot, from the compactness of its structure, undergo a diminution of its bulk, equal to that which the spongy structure of the pulmonary tissue undergoes in such cases. Consequently, when the effusion is extensive and takes place rapidly, and has diminished the bulk of the lung, by the air being expelled and the influx of the blood opposed, the pleura, which had at first contracted to a limited extent within its usual dimensions, is afterwards thrown into a multitude of narrow folds, the existence of which is perceived by the presence of parallel lines only on the surface of the inflamed pleura; but when the lung is exposed by a section, the pleura is seen doubled upon itself, or formed into folds, of the eighth of an inch in breadth, projecting in the direction of the pulmonary tissue. Fig. 2 represents acute pleurisy, which was accompanied with extensive effusion; A, A, the inflamed pleura, from which a quantity of recently effused lymph, B, B, has been removed and turned aside, in order to show the linear indications of the folds beneath, which are represented on the upper part of the figure. When the pleura, affected in this manner, is laid hold of, and stretched in the lateral direction of

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the folds, these are effaced by the separation of their opposite surfaces, and present the appearance of angular patches, C, C, of various extent, of a pale white or grey colour, showing that the pleura in these parts remained free from the inflammation which occupied all the rest of its surface. *Fig. 3* is a section of a small portion of lung, showing the manner in which the pleura is thrown into folds in the case of rapid and extensive effusion into the cavity of this membrane. A, a layer of coagulable lymph covering the pleura; B, B, the cut surface of the same, showing its thickness; C, C, folds of the pleura projecting towards the compressed substance of the lung, D. *Figs. 4* and *5* represent atrophy of the kidneys from the development of serous cysts in the substance of these organs. *Fig. 4*, A, A, the right kidney; B, the renal vein and artery; C, the ureter; D, D, D, a multitude of serous cysts covered by the proper membrane of the kidney, and occupying the cortical substance; they were equally numerous, and nearly of the same size throughout the entire substance of the kidney, and a single cyst, E, of the size of a pigeon's egg, projected from the upper extremity of the organ; a mass of fat, F, F, from half an inch to an inch in thickness, formed an envelop to the whole. *Fig. 5*, the left kidney, A, reduced to the size of a walnut; B, renal vein and artery; C, ureter; D, D, a large mass of fat occupying the place of the atrophied kidney, but much larger; E, E, a multitude of small serous cysts seen on the surface of the kidney, and which were equally numerous in its substance. The cysts are so much smaller in this than in the other kidney, that in all probability they also had undergone the process of atrophy after having effected this change in the kidney itself, and more especially in its vascular structure, the vein and artery being very much diminished in size.

PLATE II.

This plate represents that morbid condition of the liver denominated cirrhosis, which gives rise to dropsy, and which I have described as consisting in atrophy of the lobular structure of the organ, produced by the presence of a contractile fibrous tissue formed in the capsule of Glisson. *Fig. 1* is a representation of the more common appearance of cirrhosis in the early stage, when the quantity of the contractile tissue is least, as well as the obstruction to the circulation of the blood through the liver, and the effusion into the cavity of the peritoneum. A, A, the tuberiform arrangement of the lobules seen through the peritoneal covering of the liver, of a yellow rust colour; B, B, larger groups forming irregular projections; C, a portion of the peritoneum detached to shew the tuberiform arrangement more distinctly; D, D, two of the round groups of lobules separated and suspended by the constricted vessels, the corresponding portions of the liver presenting two concave depressions, E, E, in which they were lodged. *Fig. 2*, a portion of liver affected with cirrhosis, but in which the disease is much further advanced than in the former. A, A, the external surface of the liver studded with groups of lobules of various sizes of an orange yellow colour; the presence of the fibrous tissue, B, B, around these groups is very conspicuous from its great quantity, and the light grey colour which it presents; C, C, the cut surface of the liver, on which the mouths, D, D, of several veins are seen compressed. *Fig. 3*, a small portion of the liver of a cow affected with cirrhosis, which gave rise to extensive ascites: it was the only organ in the animal which presented the slightest trace of disease. Its

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bulk was considerably diminished, and its external surface, A, which was of a yellow brown colour, presented in a marked degree the tuberiform arrangement of the lobules; and on its cut surface this was rendered still more conspicuous by the difference of colour of the individual groups and the fibrous tissue by which they were separated and surrounded. B, B, B, indicates the groups of lobules, of various sizes, of a light or dark brown colour, surrounded by and subdivided into smaller ones by the fibrous tissue. The compression and obliteration of the veins, in the manner described, were also very well seen in this case, as represented in the figure at C, C, C. *Fig. 4*, a remarkable case of cirrhosis, in which the whole of the portal system of the liver was obstructed by coagulated blood, fibrine, and bile. A, A, the greater portion of the right lobe of the liver; B, C, the right branch; D, the left branch of the portal vein, from the cut orifices of which project layers of fibrine and coagulated blood, strongly impregnated with bile; E, E and F, F, large and small branches of the portal vein exposed by dissection to shew that the terminal branches, or those which enter into the composition of the lobules, as seen at G, G, G, are also distended with blood and bile. This state of the terminal veins can be observed in several of the groups of lobules on the surface of the liver, where also some of the biliary ducts are seen enlarged, H, H, H, and distended with bile. *Fig. 5*, a portion of the same liver; A, A, the external surface; B, B, the cut surface; C, C, a piece of the peritoneal covering separated to show the groups of lobules, D, and their pediculated attachment; the same tuberiform arrangement of the lobules in the substance of the liver is well marked at E, E, E, where they are surrounded by a capsule of fibrous tissue; F, F, F, the orifices of several of the portal veins, filled chiefly with fibrine of a bright yellow colour; G, one of these veins laid open, the orifices of the smaller ones, which open into it, being obstructed in the same manner; H, one of the hepatic veins also laid open to show that these vessels were not obstructed.

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Fig. 1 is a section of the same liver represented in *figs. 4* and *5* of the preceding plate, to show the large collections of fibrine and coagulated blood contained in some of the portal veins, and which in some respects resembled fungus hæmatodes; A, A and B, B, several of these collections. This figure also represents complete atrophy of many of the lobules, which are replaced by fibrous tissue, C, C, C; and others, D, D, D, which are undergoing this change, but in which their natural arrangement is still observable. *Fig. 2*, atrophy of almost the entire substance of the kidney from pressure produced by the accumulation of a sero-purulent fluid in the ureter, pelvis, and infundibula; A, A, the external covering of the kidney and condensed cellular tissue, forming a large sac, occupying the situation of this organ; B, the ureter greatly dilated; C, the cavity of the sac laid open; D, D, thin films of cortical substance, the only remaining traces of the kidney, adhering to the internal surface of the sac. *Fig. 3*, atrophy of the gall-bladder from a preternatural communication between it and the duodenum; A, A, a portion of the intestine; B, B, the ductus communis choledochus; C, the ductus hepaticus; D, the ductus cysticus; E, the gall-bladder laid open; F, a small bent probe, passed from the intestine into the gall-bladder, through the communication, at the orifice of which are lodged two small gall-stones, G. *Fig. 4*,

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atrophy of the spleen from fibrous concretions formed within, and obstructing the splenic artery; A, A, the spleen; B, trunk of the splenic artery; C and D, portions of its branches containing masses of fibrous tissue, and which are seen occupying the cavity of the vessel at E, E. The substance of the spleen, F, was composed of obliterated bloodvessels and fibrous tissue. *Fig. 5*, atrophy of the uterus and ovaries from ossification of the arteries. A, A, uterus laid open; B, B, Fallopian tubes; C, C, ovaries; D, D, D, the principal arteries, and several of their smaller branches completely ossified and nearly impervious; the substance of the uterus, E, containing a multitude of small arteries in the same state; a tumour, F, composed of dilated veins and cellulo-fibrous tissue, occupying the fundus of the uterus.

P L A T E I V .

Fig. 1, atrophy of the convolutions of the brain in general paralysis of the insane, affecting both hemispheres, and more extensive in this case on the left than on the right side. A, A, right hemisphere; B, B, left hemisphere, the venous trunks of which are gorged with blood; C, C, C and D, D, D, a great number of convolutions on both hemispheres atrophied, and their places occupied by serosity accumulated beneath the pia mater; E, E, a portion of the pia mater turned aside, in order to expose the atrophied convolutions, several of which, F, F, have undergone a diminution of bulk, chiefly in thickness; others, G, G, both in thickness and depth, and have almost entirely disappeared, thus leaving deep irregular hollows, in which the effused serosity was contained. *Fig. 2*, atrophy of the left anterior pyramidal and olivary bodies, the left half of the pons and crus of the brain from extensive destruction of the left corpus striatum; A, A, cerebellum; B, left, C, right crus cerebri; D, pons Varolii; E, medulla oblongata; F, right, G, left pyramidal body; H, right, K, left olivary body. *Fig. 3*, atrophy of the left lung from obliteration of the principal bronchus by a mass of tuberculated glands, from a monkey. A, A, right lung; B, B, left lung; C, C, the diseased glands surrounding the obliterated bronchus from the bifurcation of the trachea D, to within a short distance of the termination of the former, E, in the lung; F, F, the cut orifices of the compressed and obliterated bronchus; G, G, G, G, a great number of miliary tubercles occupying both lungs; H, H, groups of lobules affected with emphysema, and confined to the right lung. *Fig. 4*, a peculiar diseased state of the chord and pons Varolii, accompanied with atrophy of the discoloured portions; A, right crus, B, left crus cerebri; C, pons Varolii; D, medulla oblongata; E, E, medulla spinalis; F, F, isolated points of the pons Varolii, of a yellowish brown colour; G, G, G, patches of the same kind on the spinal chord, all of them occupying the medullary substance, which was very hard, semi-transparent, and atrophied. The atrophy was more conspicuous in some points than in others, and is particularly well seen in the figure at H, where it affects a portion of the right olivary body; K, softening of a portion of the chord. *Fig. 4*, A and B, represents transverse sections of the chord, to show that the discolouration commences on the surface of the white, and extends inwards to the grey substance; A, the brown discolouration which has extended to the grey substance on one side of the chord, a; and is confined to the white substance on the other side, b. Similar appearances are represented in the section B, a, b.

Plate I.

Fig. 1.



Fig. 2.

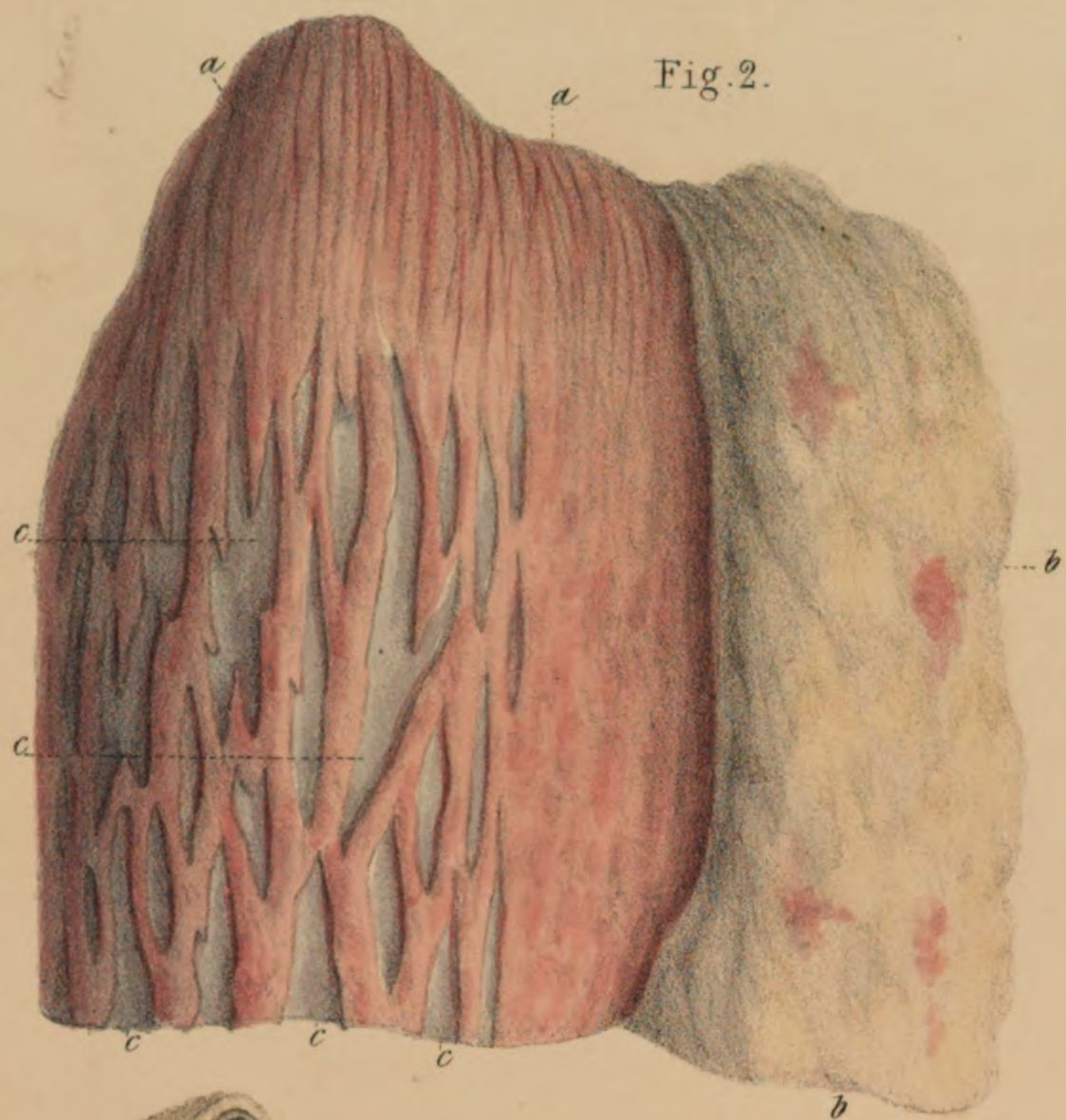


Fig. 3.

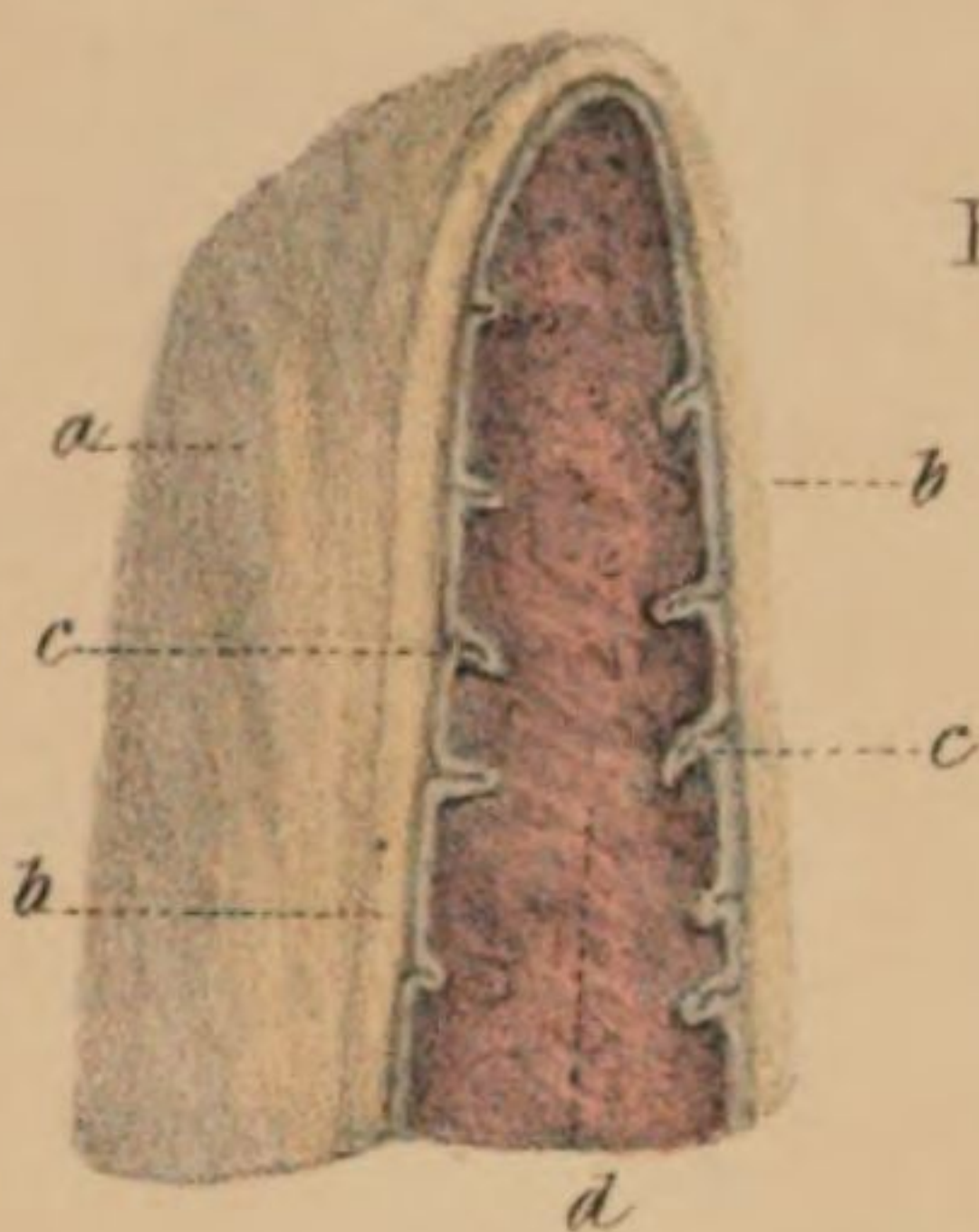
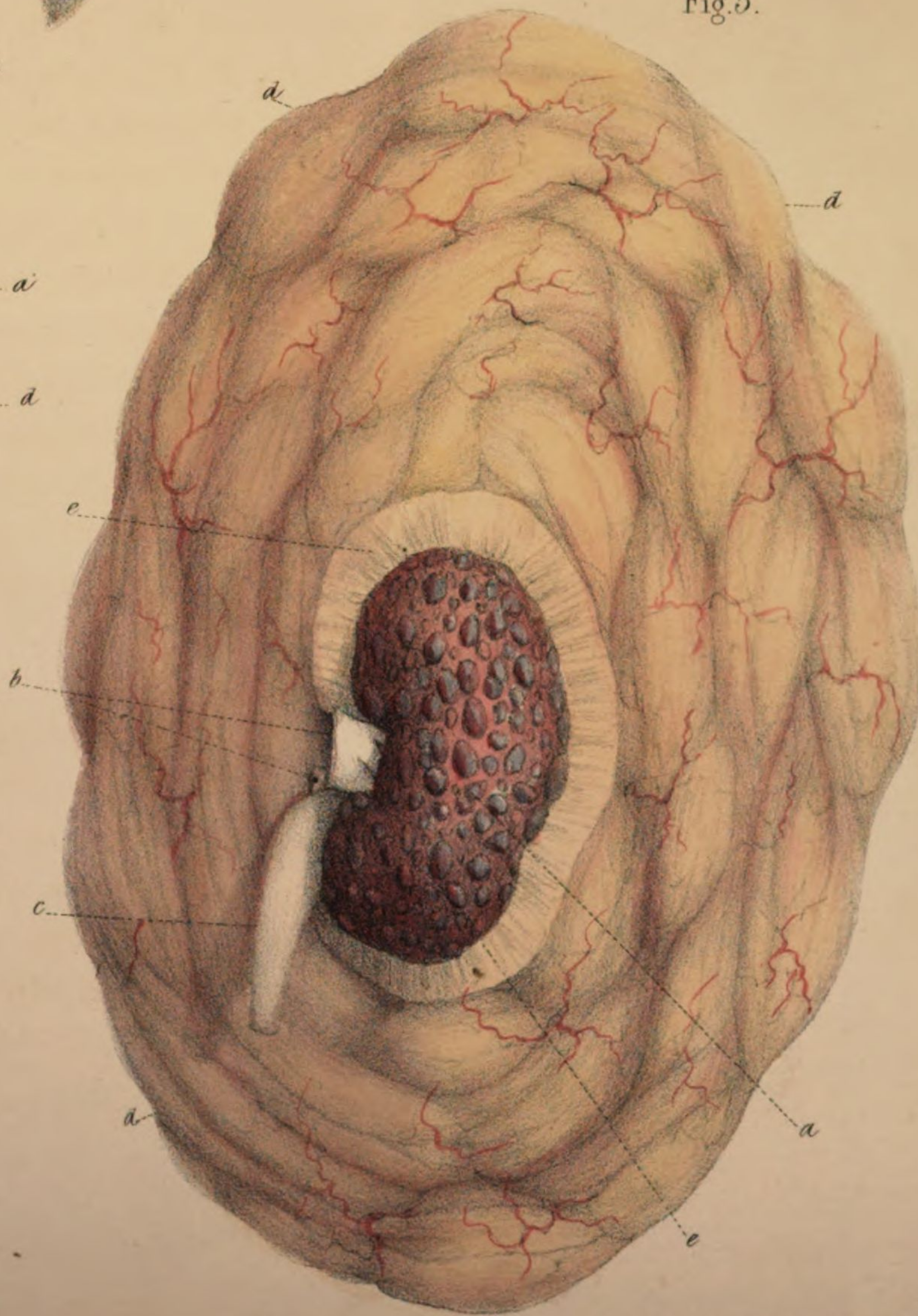


Fig. 4.



Fig. 5.



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Plate II.

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Wells & Co

Plate II.

Fig. 2.



Fig. 1.

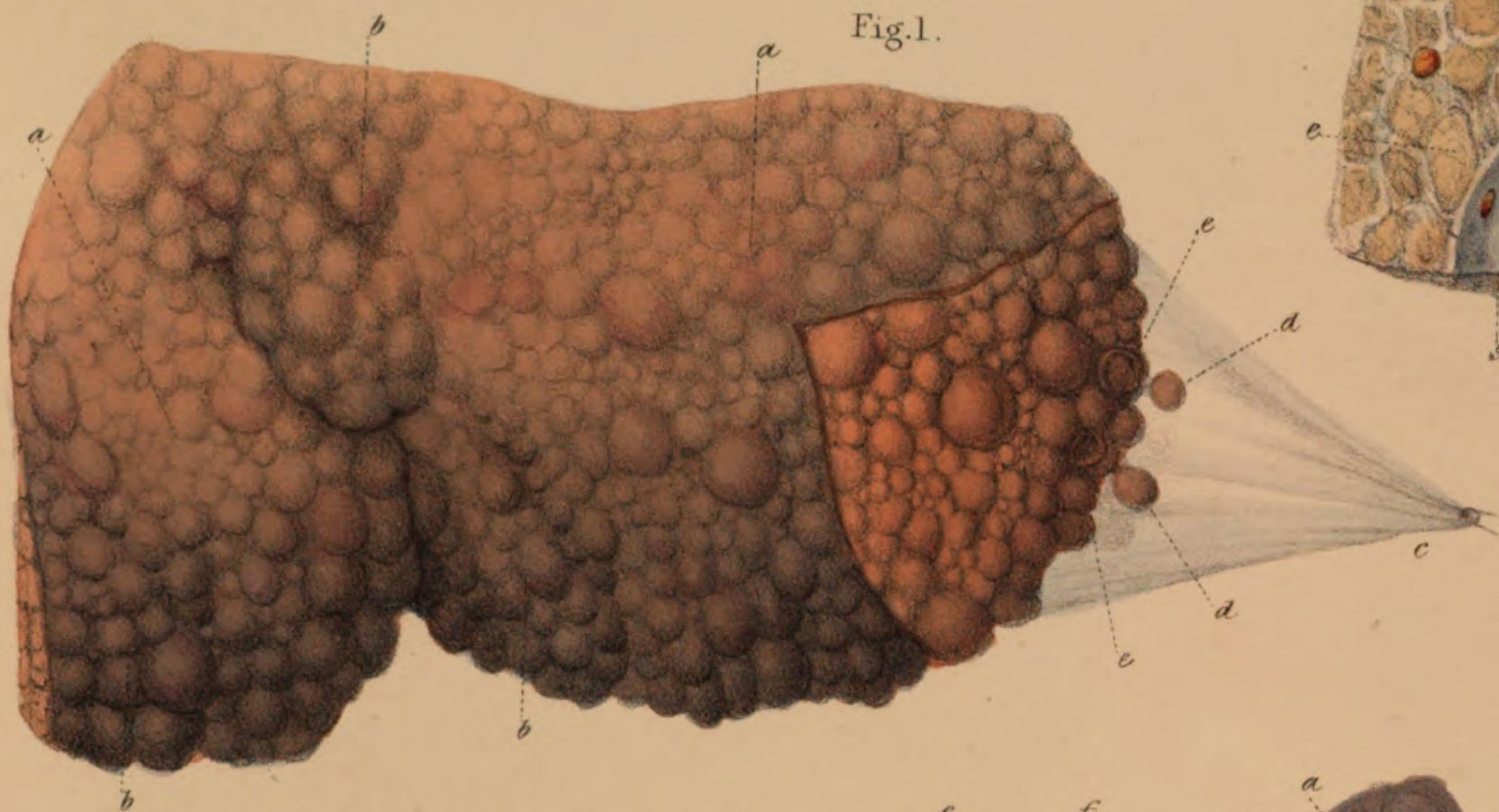


Fig. 5.

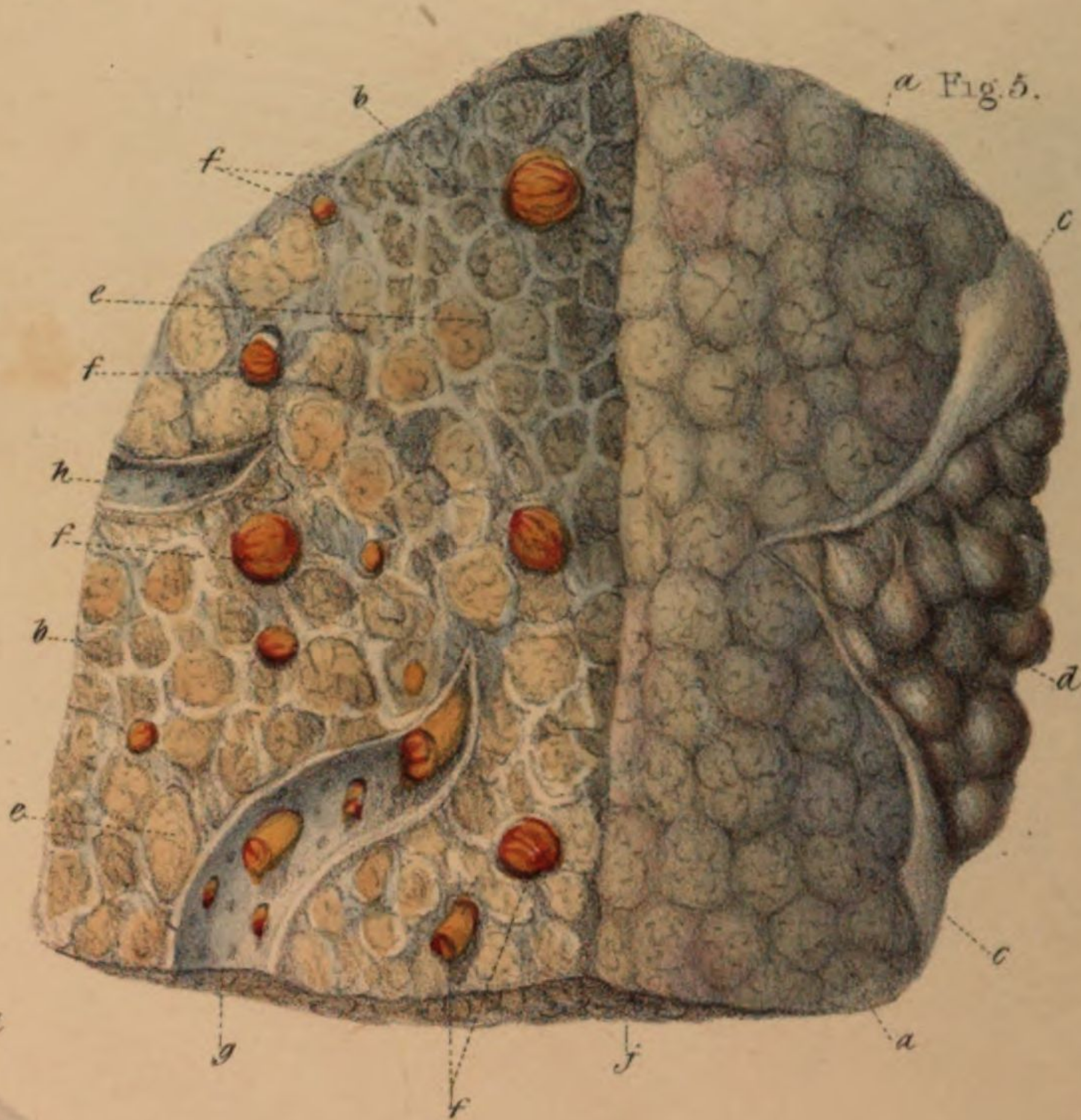


Fig. 3.

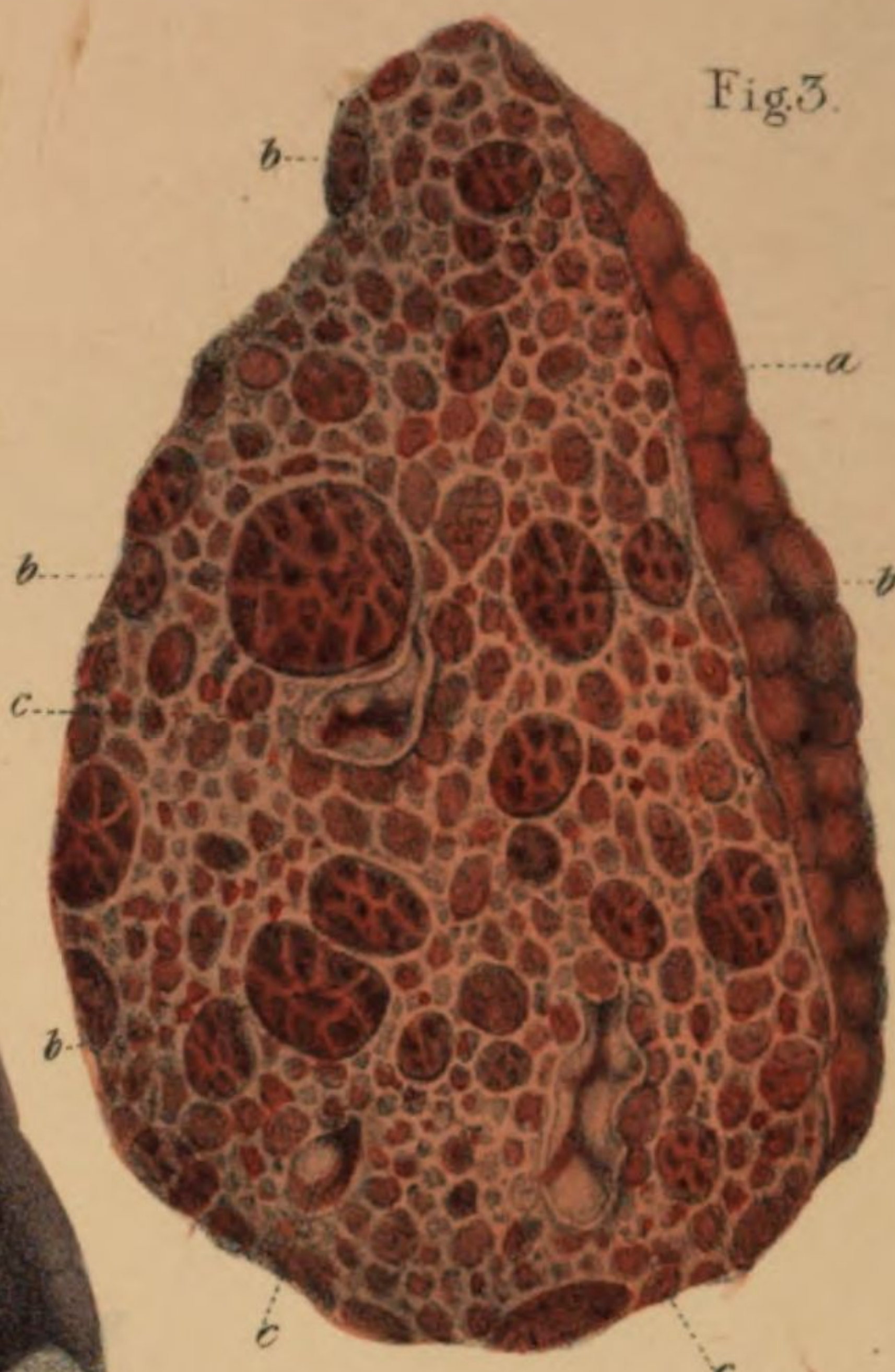


Fig. 4.

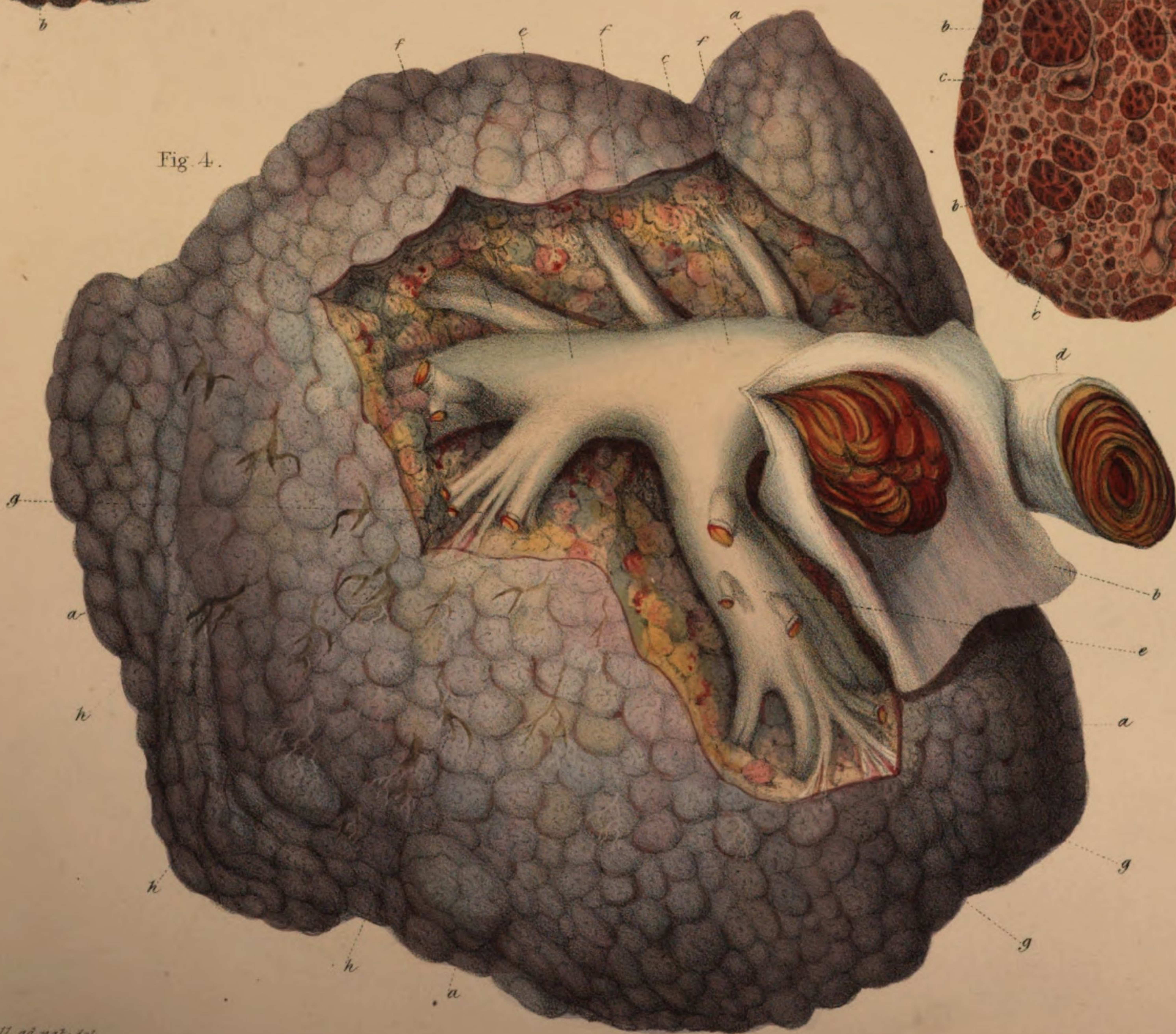


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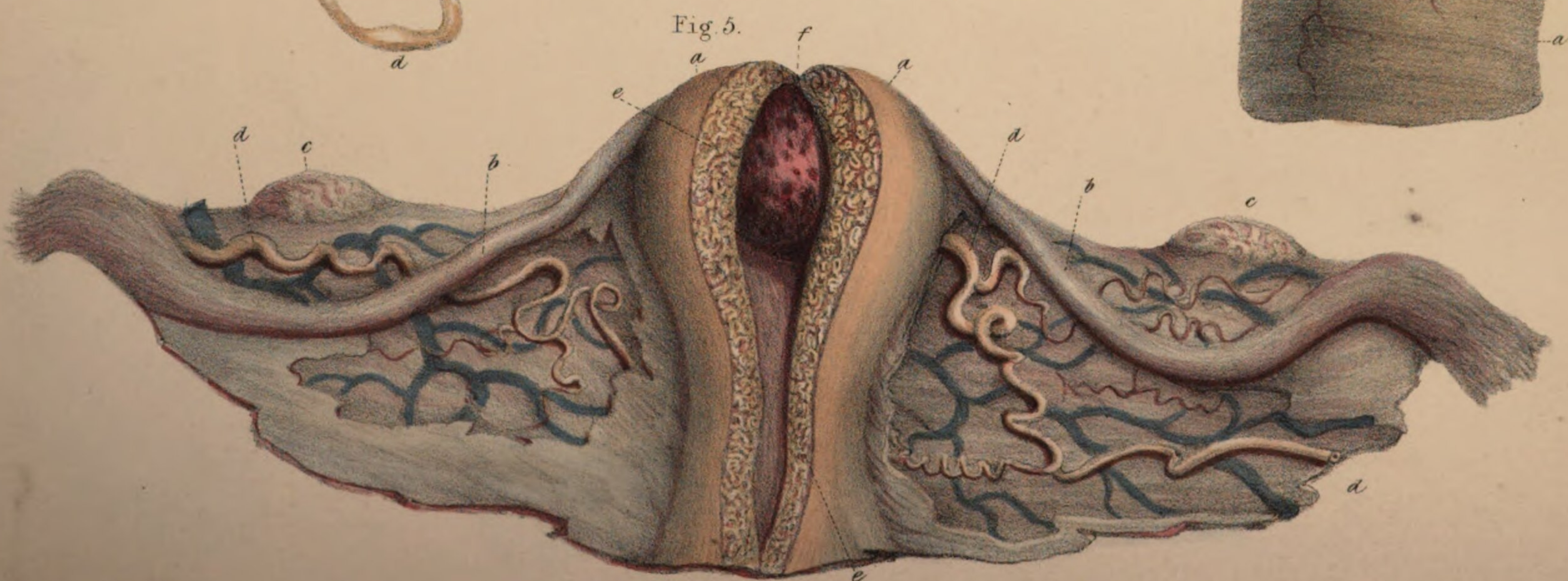
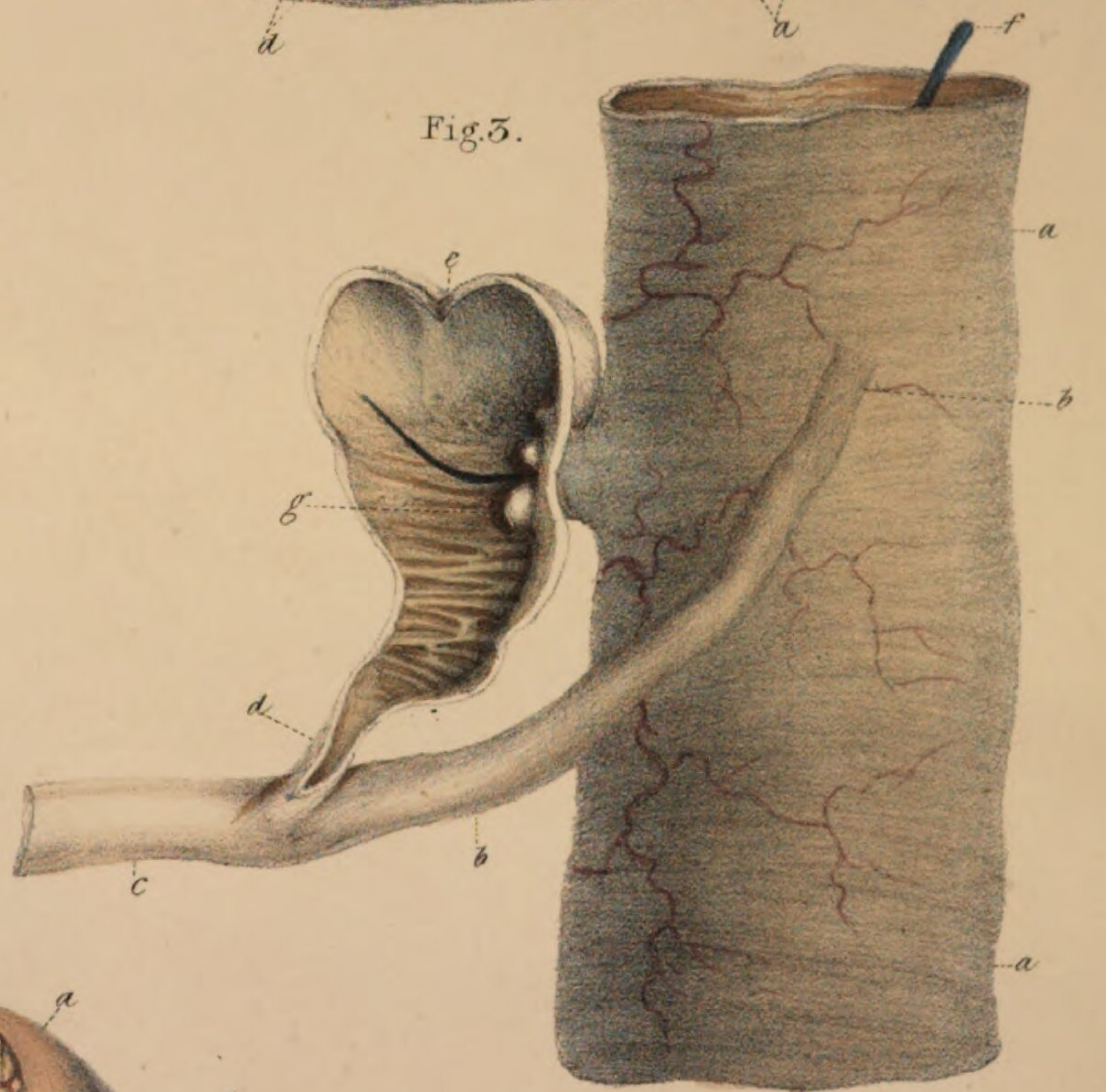
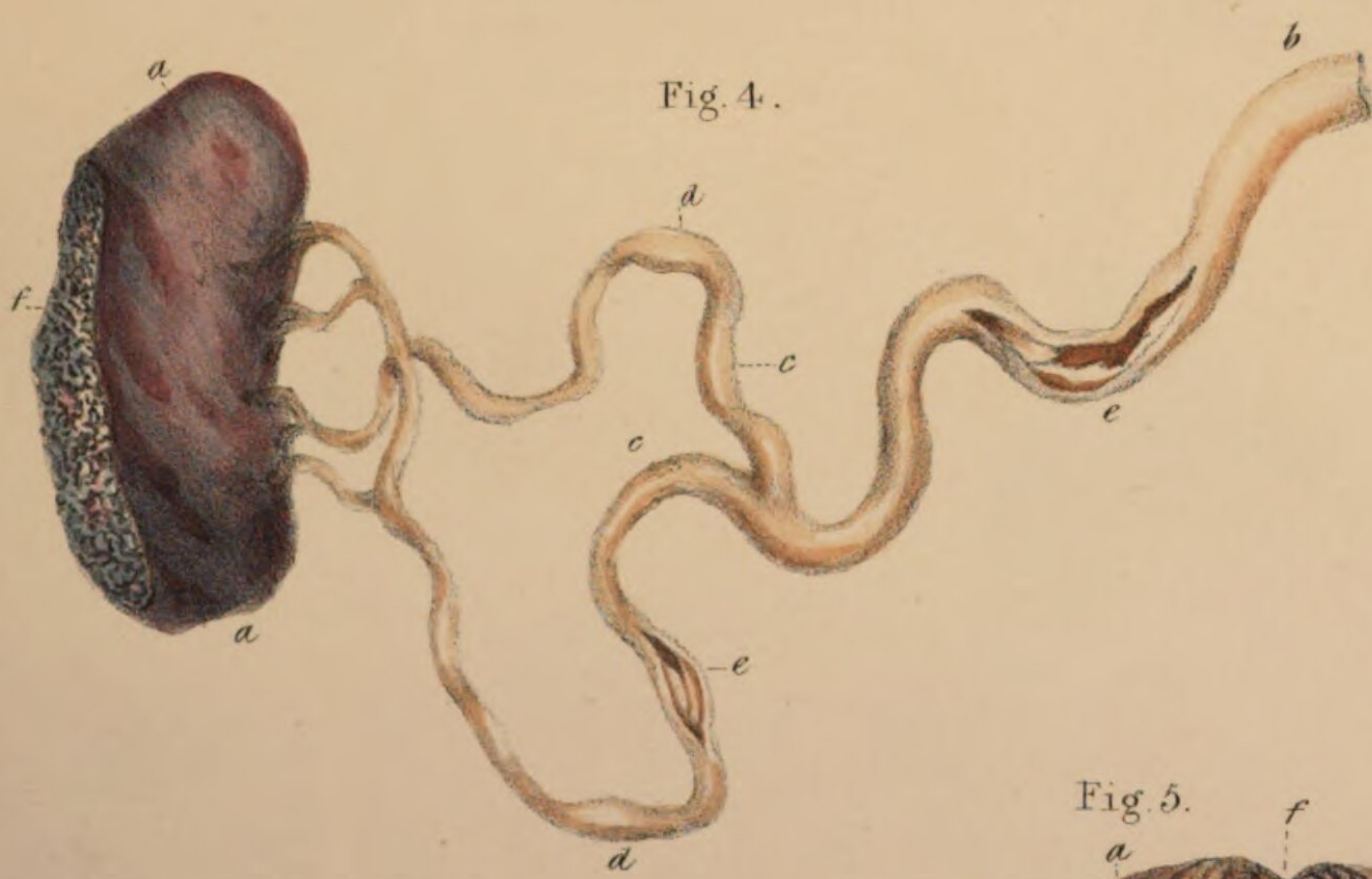
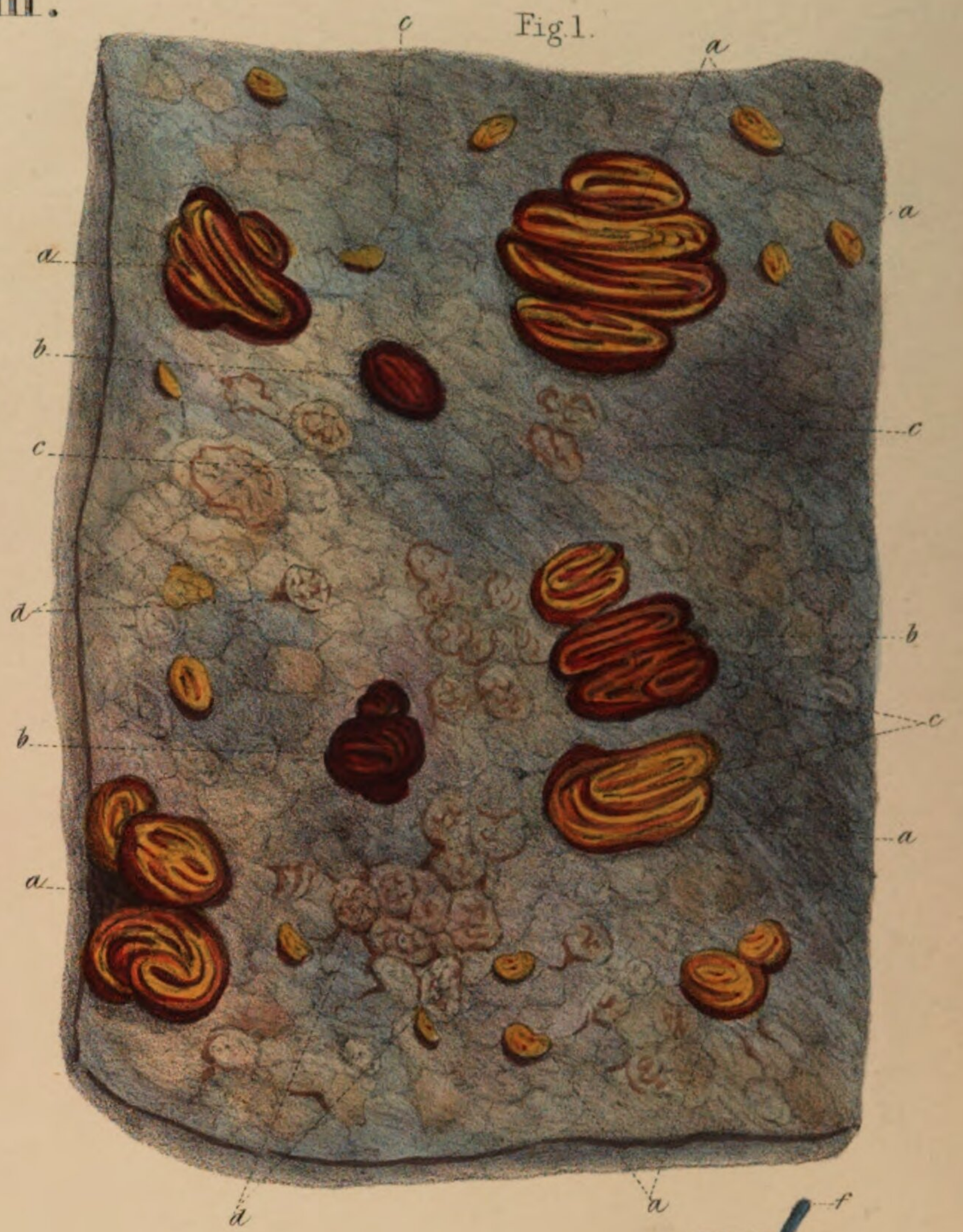


Plate IV.

Fig. 1.

Fig. 4.

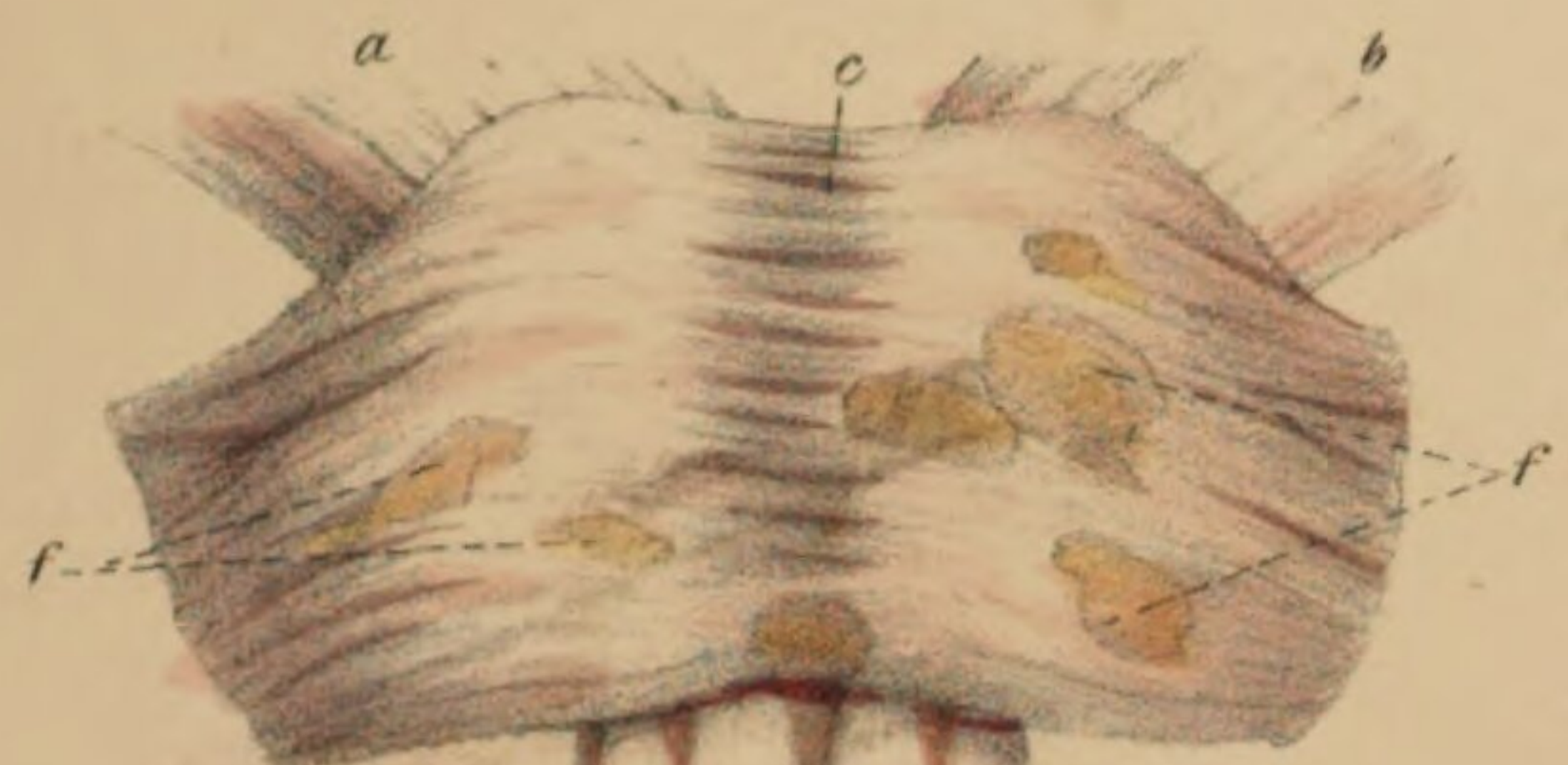
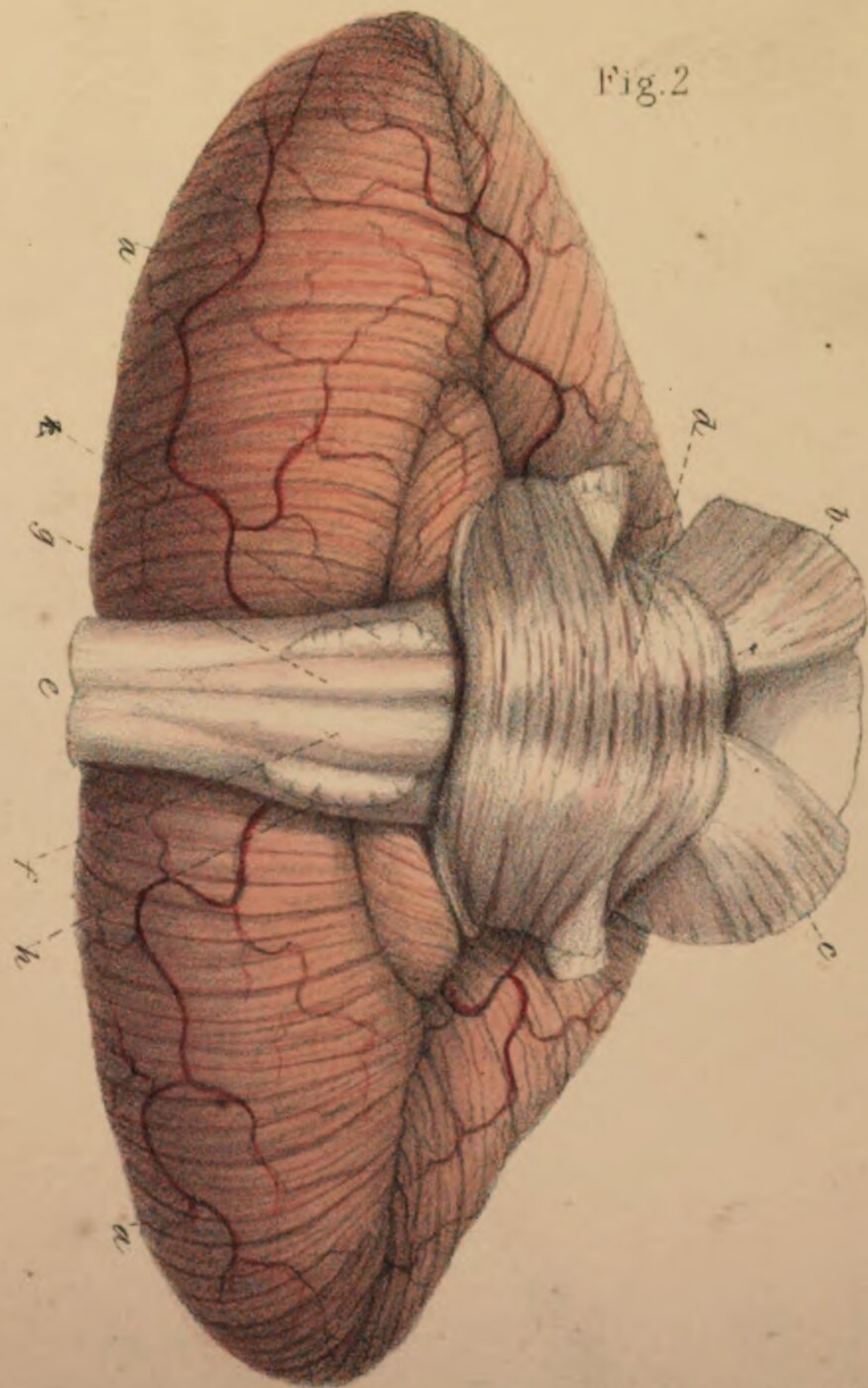


Fig. 4.



Fig. 3.

Fig. 2.



HYPERBOLY

The first question which arises in the mind of the student is, what is the meaning of the word? It is a word of Greek origin, and signifies a curve which is not a circle, but which approaches a circle as the radius increases. It is a curve which is not a circle, but which approaches a circle as the radius increases. It is a curve which is not a circle, but which approaches a circle as the radius increases.

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THE solid materials which enter into the composition of the healthy structure of the body frequently undergo remarkable modifications in quantity. These modifications consist either in an increase or diminution of quantity, and become conspicuous in the deviations to which they give rise in the normal bulk or dimensions of organs and tissues. As both of them imply simply a relative change of bulk in reference to healthy composition, they may be regarded as essentially dependent on a corresponding increase or diminution of the nutritive function, and therefore the former has been termed Hypertrophy, the latter Atrophy. We shall treat of Hypertrophy only in the present fasciculus.

Considered in a general point of view, and as the result of the exercise—anormal merely in degree—of a physiological act, hypertrophy is certainly one of the least complicated of the numerous changes which have been included among the pathological conditions of the solids. It constitutes, however, in some tissues, more especially the muscular, perhaps more frequently a physiological than a pathological state. It belongs strictly to the former as regards its origin and organic conditions or structural relationship, and can hardly be said to come within the denomination of the latter until it interferes with the regular and effective accomplishment of a function. This latter circumstance, which, in some organs, is of frequent occurrence and productive of severe and often fatal diseases, has been fully appreciated by modern pathologists, and has rendered the study of hypertrophy one of great interest and importance. We shall therefore treat more especially of hypertrophy when occurring as a pathological condition, giving only a general view of those forms which are unaccompanied by any derangement of function, but which, nevertheless, serve to illustrate its nature in general, and the mode of operation of the causes by which it is most frequently produced.

Nature of Hypertrophy.—That the increased anormal development of an organ or tissue, denominated hypertrophy, depends essentially on an excess of the nutritive function, appears to be sufficiently demonstrated by the presence, on the one hand, of an increase of bulk, and the absence, on the other, of any adventitious solid or fluid substance. The organisation and structure remaining unaltered is also further evidence that the increase of bulk is owing to a superabundant deposition of the natural solid constituents of the affected organ, from an excess of the nutritive function. Our ignorance of the manner in which the vital and complicated function of nutrition is performed, forbids any attempt to illustrate more satisfactorily the nature of hyper-

[*Hypertrophy*, 1.]

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trophy. It would be altogether hypothetical to maintain that hypertrophy is the consequence of an accumulation of the nutritive materials, from a diminished exercise of the act of decomposition, or that the increase of bulk is effected by a numerical increase of the molecular composition. It would appear to be more consistent with the present state of our knowledge to say, that hypertrophy is the consequence of an excess of the nutritive function, because we thus express a fact which is in accordance not only with the nature of the change produced, viz. an excess of bulk, but also with the general law of organic development. As an active and vigorous state of the circulation, and consequently a copious supply of blood, would seem to be conditions necessary to the full development of the body as a whole, it is reasonable to suppose that similar conditions are required for the production of hypertrophy, or an increased development of an individual tissue or organ. That such are the conditions on which the production of hypertrophy depends is most satisfactorily demonstrated by a consideration of the causes under the influence of which it is so frequently observed to occur both in the voluntary and involuntary muscles. The prodigious development and power of the muscles of the superior extremities of the blacksmith, and of the inferior extremities of the stage-dancer, are striking examples of hypertrophy occurring under the influence of the frequent and increased exercise of a function, the effect of which is a corresponding increase of nutrition and of development of the muscular tissue. The anormal development of the muscles of involuntary motion, as those of the heart, urinary bladder, and intestines, is likewise often precisely of a similar nature, an increased exercise of the muscular power of these organs having been excited and kept up to overcome a mechanical obstacle to the free passage of their respective contents.

That an increased supply of blood is necessary to the production of hypertrophy, appears to be put beyond doubt by the state in which the vessels are found through which this fluid is immediately conveyed to the affected organ. They are often much more capacious, that is to say, larger than usual, having acquired an increase of size corresponding with the bulk of the organ to which they have supplied the superabundant materials of its nutrition. This is remarkably conspicuous in hypertrophy of one kidney with atrophy of the other, and is also frequently observed in cases of great hypertrophy of the muscles of voluntary and involuntary motion. This state of the bloodvessels confirms the evidence already adduced in support of the opinion that hypertrophy is the result of an excess of the entire process of nutrition as accomplished in the solids, and not of a diminished activity of the act of decomposition; or, as it has also been supposed, of an increased activity of the process of assimilation and a more than usual plasticity of the blood.

The conclusion at which we have arrived in these general considerations on the nature of hypertrophy merits due consideration, not only as it involves the principle on which the successful treatment of this pathological state is founded, but also as it establishes a law in direct opposition to the doctrine that this change is frequently the primary element of accidental formations which have no analogy in the economy. That hypertrophy of the cellular tissue can form a necessary condition of a heterologous product such as carcinoma, or can, *per se*, be transformed into such, is a statement which the nature of this change, essentially physiological in its vital and organic conditions, shows to be equally fallacious and unphilosophical.

Origin and Causes of Hypertrophy.—Hypertrophy occurs under circumstances so very various and so different in kind, that a correct estimate of its nature, in individual

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cases, could not be obtained without a previous knowledge of the causes in which it originates. The most remarkable difference in this respect is observed in that form of hypertrophy from arrested or imperfect development, in consequence of which an organ retains after birth that relative condition of bulk which was necessary to its accomplishing a function peculiar to the foetal state of existence. This is observed sometimes to occur in the heart, an organ which, before birth, is in that state of hypertrophy called concentric. The thickness of the walls compared with the capacity of the ventricles of the heart in the foetus, is nearly twice that of the walls compared with the capacity of the ventricles of this organ in the adult. This relative disproportion between the walls and cavities of the foetal heart is conspicuous in infancy and childhood; but during the regular development of the body, it gradually merges into those proportions of capacity and bulk which are regarded as indicating, in these respects, the normal state of the heart. If, therefore, the changes which should take place in the regular development of the heart after birth are prevented, it is obvious that the permanence of the state of the foetal heart which we have described, must, under new and so very different conditions of life, constitute a pathological state, a state of concentric hypertrophy, and give rise at an early period to those derangements of function which follow as the consequences of this disease; and that such is the origin of some cases of this form of hypertrophy is proved by a careful examination of their early history, and of the organ itself after death.

It is worthy of remark that what may be called *congenital hypertrophy*, may be accompanied either by an increase or a diminution of the bulk of the heart. I have not, however, found that the bulk of this organ, compared with the size of the body in such cases, was ever very much increased. I have met with it oftener of the normal bulk, and once so small, in the body of a female above the age of forty, that it did not exceed the dimensions which it usually presents at the age of twelve or thirteen. (See Plate II. *figs.* 6 and 7.) The persistence after birth of the foetal state of the liver, as to the greater size of the left lobe, is occasionally met with, and might, possibly, interfere with the process of digestion; and the thymus gland has been found to retain for a considerable time after birth its original dimensions, and to give rise to distressing and even fatal consequences.

Of the causes which manifest their effects in the production of hypertrophy, some exercise a local, others a general influence. The more remarkable of the former appear to reside in a predisposition or peculiar organization of the individual, in which the function of nutrition in general, rather than any one of its special acts, is so modified as to give rise to an increased but imperfect development of various organs and tissues of the body. Such a state is observed in those persons who are said to have a scrofulous constitution, and in whom the liver, bones, lymphatic glands, sometimes the brain, and frequently the upper lip, are more or less obviously enlarged.

A preternatural development of the adipose tissue, sometimes carried to an enormous extent, is likewise observed to occur under circumstances of predisposition, inasmuch as the habits and modes of life of the persons affected may not differ in kind or degree from those of other persons who are not so affected. Indeed, polysarcia will take place in some persons, and proceed to a great extent, in spite of the most abstemious habits, exercise, and other means employed to prevent it.

Among the local causes of hypertrophy may be enumerated the three following:—
1st, the frequent and increased action of an organ in the normal exercise of its function;

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2d, the existence of a mechanical obstacle to the accomplishment of the function of an organ; 3d, the long-continued influence of a morbid stimulus. Hypertrophy under the influence of one or other of these causes occurs in almost all the organs and tissues of the body. It may affect an entire organ, or one or more of its elementary tissues, exist as the only perceptible lesion, or be accompanied with other diseased states.

1. *Hypertrophy from the frequent and increased action of an organ in the normal exercise of its function.*—It is this form of hypertrophy which occurs in the voluntary muscles, more especially in those of the extremities, which we have already alluded to, and is so obvious as to require no further illustration. Although it does not constitute a pathological condition, yet it cannot take place to a great extent in one region of the body without exercising a marked influence in the development of some other region, thereby illustrating the law of regular organic development. Thus, the superior extremities of celebrated *danseuses*, when compared with the powerfully developed inferior, often appear as if emaciated; and the legs of the *drayman* and others brought up to similar occupations, are sometimes so slender that they seem hardly to possess sufficient strength to sustain the weight of the broad chest and brawny shoulders of these individuals.

But a more important variety of this form of hypertrophy occurs in some involuntary muscles and double organs. Great hypertrophy of the heart is by no means uncommon, where no perceptible mechanical obstacle exists to the circulation of the blood, either in the heart or great bloodvessels, and where no local morbid stimulus can be traced to have contributed to produce this change. In cases of this kind, an excess of nutrition appears to be the consequence of an increased exercise of the heart, induced, in a manner which has not yet been satisfactorily explained, through the agency of certain diseased states of other organs, particularly of the brain and nervous system, and various affections of the mind. The increase of bulk which takes place in one lung when the function of the other has been suppressed by a pleuritic effusion, or by an accidental membranous tissue compressing and fixing it to the spine; or that of one kidney when the other has been destroyed by disease, are beautiful and interesting examples of hypertrophy from a physiological increase of function, called into operation by the wants of the economy for the maintenance of life. The increased development of the arteries and veins, by means of which a collateral circulation is established for the preservation of a limb or even of life itself, as in cases of ligature of the femoral artery, and obliteration of the upper portion of the abdominal cava, are no less interesting and important examples of hypertrophy of a similar kind: nor should we, perhaps, consider as essentially different from the former, the increase of bulk and capacity which, as a purely physiological act, takes place in the uterus during the development of the impregnated ovum, as a necessary condition to the formation and growth of a separate and individual existence. An increase of bulk of the mammary glands rarely fails to take place after parturition, and an extraordinary development of them sometimes follows the exercise of their special function from frequent and long-continued lactation. That one hemisphere of the brain, the other being in a state of congenital atrophy, acquires greater dimensions than in ordinary circumstances, is probable, although it has not, so far as I know, been satisfactorily ascertained. It is asserted by Phrenologists that particular portions of the brain acquire an increase of bulk under the influence of a more early and prolonged exercise of some of the special faculties of the mind, or a greater indulgence of certain passions than others.

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2. *Hypertrophy from the existence of a mechanical obstacle to the accomplishment of the function of an organ.*—Hypertrophy from a mechanical obstruction to the accomplishment of a function is not only of frequent occurrence, but is carried to an extent which is never observed under any other circumstances. The most common and obvious cause of hypertrophy of all hollow organs is a prolonged obstruction to the transmission of their contents. Thus, perhaps the most frequent, and certainly the most effective causes of hypertrophy of the heart are contraction of the aortic, pulmonary, and auriculo-ventricular orifices, produced by various diseases of the valves, and the presence of accidental formations connected with these parts; smallness of the aorta; diminished elasticity of the middle coat of the aorta or pulmonary artery; dilatation and the consequent non-occlusion of the auriculo-ventricular orifices; diseases of the chest which impede for a considerable time the circulation of the blood through the lungs, more especially chronic bronchitis, emphysema, &c. Contraction of the pyloric orifice of the stomach and of various portions of the intestinal canal, from the gradual development of accidental formations within their parietes, or projecting from their internal surface, or from the cicatrization of ulcers, more especially of the latter, are by far the most frequent causes of hypertrophy of the muscular coat of these organs, and which, in consequence of the accumulation of their contents, is always accompanied by an increase of capacity or dilatation. Diseases of the prostate gland and stricture of the urethra, which obstruct for a long time the passage of the urine, and occasion an accumulation of this fluid in the bladder, are followed by hypertrophy and dilatation of this organ to a greater extent than is observed under any other circumstances. From the same causes also, but more frequently from diseases of the uterus, ovaries, and bladder, which produce compression or obliteration of the orifices or inferior extremities of the ureters, these canals undergo great increase of bulk, both in the thickness of their walls and in their capacity. Obstruction at or near the orifices of mucous and sebaceous follicles and of all excretory ducts, occasioned by the presence of what are called concretions, and accidental formations situated within or external to them, is perhaps the sole cause of the great dilatation which some of them occasionally present, more particularly the hepatic and pancreatic. In cases of great dilatation of the biliary ducts, occasioned by a large concretion lodged in the ductus communis choledochus at its duodenal extremity, there is always observed a remarkable development of what appears to be a muscular tissue, as it resembles very much that of the bronchi under similar circumstances. It occupies the situation of the cellular tissue external to the mucous membrane, and presents a marked reticular arrangement in the common, cystic and hepatic ducts, and in the gall-bladder, where it likewise sometimes assumes a stellated arrangement. In both cases the bundles of fibres are as conspicuous as in dilatation with hypertrophy of the bronchi. In like manner, a mechanical obstruction to the passage of the blood through the veins, and of the lymph and chyle through the lymphatics and lacteals, is sometimes followed by considerable hypertrophy and dilatation of these vessels.

The most remarkable case of dilatation of the lymphatics which I have seen, occurred in a young man about twenty-six years of age. My friend, M. Amussat of Paris, was called to the patient, who the day before had been seized with severe pain in the abdomen, followed by frequent vomiting. These symptoms, and the presence of two swellings, one in each groin, nearly as large as an orange, left no doubt that the patient was labouring under the effects of strangulated hernia, but the state of prostration was such that reduction by an operation was not attempted. On examining the patient

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after death, the only remarkable circumstance observed was enormous dilatation of the lymphatics from both groins upwards, including the thoracic duct. The two swellings situated in the groins, and which at an early age of the patient had been treated as a case of *double hernia*, (for we afterward learned that he had worn a double truss from his boyhood,) were found to be produced by great dilatation of the lymphatics of the inguinal glands. When cut into, instead of having a compact structure, they presented the appearance of a coarse sponge, from the size of all these vessels being increased, the most of them presenting from one to three lines in diameter. All the lymphatics of the pelvic and lumbar regions presented the same alteration in a still more remarkable degree. None of them were less than two, many of them from three to four lines, and the thoracic duct was from six to eight lines in diameter. As no obstacle was found in the course or at the termination of the thoracic duct to account for the dilatation of the lymphatics in this singular case, and as these vessels had undergone no other perceptible change, I am disposed to consider it as an example of *malformation* of these vessels, more especially when these facts are connected with the circumstance furnished by the history of the case, viz. the existence, at an early period of life, of the enlarged state of the inguinal glands, the nature of which I have explained. (See Plate IV. *fig.* 4.)

3. *Hypertrophy from the long-continued influence of a morbid stimulus.*—Neither of the causes which I have described exercises so general an influence in the production of hypertrophy, as that modification of function which succeeds to the operation of a morbid stimulus. A state of irritation or chronic inflammation thus produced is by far the most frequent cause of hypertrophy of the mucous, cutaneous, cellular, fibrous, and osseous tissues, and glandular organs; nor is it an unfrequent occurrence in the involuntary muscles, and is occasionally met with in the brain, the nerves, and their ganglia. It is necessary, however, to observe, that an increase of bulk of these tissues and organs from this cause, is not only apt to be confounded with, but is sometimes not to be distinguished from, other pathological states to which they are subject. Thus, serous and albuminous infiltrations of the skin, mucous membranes, lymphatic glands, and perhaps also of the substance of the brain, sometimes occasion an increase of bulk which would readily be confounded with hypertrophy of these parts, were they not subjected to minute and careful examination. The organization of coagulable lymph effused into the cellular tissue of every part of the body, and also on the free surface of serous membranes, is a common source of error in this respect, in consequence of the organized lymph assuming all the physical characters of both, and thus rendering it impossible in most cases to ascertain whether the increase of bulk in the one, and of thickness in the other, be owing to hypertrophy or to the presence of an analogous new formation.

From the remarks made on the nature of hypertrophy on entering upon the consideration of this subject, it would appear that irritation kept up for a length of time in a tissue gives rise to this modification of nutrition, from the well-known fact that the capillary circulation is always increased under such circumstances. That hypertrophy is produced from this cause and under such circumstances is put beyond all doubt by daily observation. Examples are common in the cutaneous and mucous tissues, in chronic ulceration of both, and in irritation and chronic inflammation of the latter, especially of the mucous membrane of the air-passages, digestive canal, and urinary bladder. But it is not only as a whole that the skin and mucous membrane are increased in thickness; their anatomical elements also sometimes acquire a remarkable development, and render

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conspicuous to the eye the peculiarity of their structure, which before was either not at all perceptible or of very difficult demonstration. Andral has given a very interesting example of hypertrophy of this kind of the skin of the right leg, which, thirteen years before, had been the seat of ulceration, and had ultimately acquired a great increase of bulk. On examining the thickened skin from within outwards, it was found to consist, 1st, of the *cutis vera*, much thickened; 2d, of the *papillary tissue*, which, distinguished with difficulty from the former in the natural state, was in many points much enlarged and elongated; 3d, of three layers situated between the latter and the epidermis, differing from each other in colour, consistence, and structure. The first of these layers, covering the papillary tissue, was considered to be analogous to that which Dutrochet has called the *epidermic* layer of the papillæ; over this again was situated another layer of a dark grey or brown colour, having a reticular structure, occupying the place of, and obviously analogous to, the *rete mucosum*; and lastly, a much thicker and harder layer than the two former, which, existing only in a rudimentary state in man, presents various degrees of development in animals, and contributes to the formation of shell and horn.

A similar development of the elementary structure of the mucous membrane is frequently observed to take place in consequence of chronic inflammation. The follicles of the intestines, especially the glandulæ solitariæ and agminatæ, are often affected with hypertrophy. The former of these glands, the situation of which is not perceptible to the naked eye, become as large as pins' heads or hemp-seed, and project above the surface of the mucous membrane; and the latter, which in the natural state are on a level with the mucous surface, are sometimes raised above it more than a line. The villi also undergo great enlargement, and become much elongated from the same cause. Besides, some of the mucous membranes which present no villous structure in the normal state, acquire this structure when hypertrophied. This has been observed in the mucous membrane of the bronchi and urinary bladder, a remarkable example of which is represented in Plate I. *fig. 2*.

Hypertrophy of the cellular tissue from irritation or chronic inflammation is considered the most common of all. Occurring in every part of the body, it is, however, most frequent and greatest in degree where it is called the subcutaneous and submucous tissue, and is not uncommon, though less considerable, where it forms an anatomical element of compound organs. Serous membranes do not appear to acquire an increase of thickness from hypertrophy; nor do fibrous membranes or tendons undergo this change in a marked degree. In dilatation of the arteries and veins the middle coat is often much thickened, in many cases, obviously from a pathological condition similar to that which gives rise to hypertrophy in other analogous tissues. Thus, in such cases we find the cellular tissue situated between the middle coat and lining membrane of the vessels thickened; this membrane opaque and of a pale-straw colour, and the middle coat much less elastic than in the natural state, or altogether deprived of this property. This is almost always the pathological state of the walls of an artery in dilatation, whether occupying its whole circumference, or a portion of it in the form of a circumscribed aneurismal tumour. Nor is it confined to the arteries: it occurs in the heart itself, sometimes being limited to a small portion of its walls, at others affecting a great part of one ventricle. The first perceptible lesion in aneurism of a portion or of the entire walls of one ventricle of the heart, (of which I have met with several examples, and always in the left ventricle,) is in every respect similar to that which so often precedes the formation

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of aneurism of the arteries. The serous membrane lining the internal surface of the ventricle, presents within a circumscribed space, varying from a quarter of an inch to one or two inches in breadth,—or, as in two examples which I have met with, from one-half to two-thirds of its entire extent,—a pale-straw colour; it has become opaque, is closely united to the cellular tissue beneath it, which presents the same colour, and is considerably thickened. Occupying the situation in which these changes are perceived, and sometimes nearly to the same extent, are one, two, three or more depressions, cavities, or sacs. These are lined by the serous membrane and cellular tissue, which appear as if they had been gradually extended and pushed outwards, carrying before them and ultimately causing atrophy of the muscular substance of the heart, the only change which it suffers in such cases. The formation of the cavity or aneurismal sac depends obviously on two causes; 1, the loss of resistance in the serous membrane from disease; 2, the muscular force of the heart, under the influence of which and through the medium of the blood the diseased membrane must yield, be forced outwards, and by compression interrupt the capillary circulation in the muscular tissue, which will become atrophied by interstitial absorption, and thus favour the continual operation of the mechanical cause. While this process is going on, the serous membrane and its accompanying condensed cellular tissue are gradually elongated, and form a smooth though sometimes unequal covering to the sac, the fundus of which is sometimes not larger than the opening by which it communicates with the cavity of the ventricle, at other times much larger, in which case only it has been found to contain coagula or a quantity of fibrine.

Besides the morbid conditions of the internal lining membrane and its sub-cellular tissue which give rise to circumscribed aneurism of the heart, there are others, connected however with these, and having apparently the same origin, and which I have always found to accompany the uniform dilatation of the ventricle in the direction of the affected walls. In the cases in which I have observed this form of dilatation, the opposite surfaces of the pericardium were intimately united by firm cellular tissue, the consequence of a previous attack of pericarditis. The lining membrane of the heart and its sub-cellular tissue presented the same appearances as in circumscribed aneurism; but there was, besides, a greater or less quantity of cellulo-fibrous tissue, occupying the situation of the muscular substance of the ventricle, sometimes throughout the whole thickness of its walls. It is in the situation of this tissue, as I have said, that the dilatation takes place. That this as well as the circumscribed form of dilatation originates in inflammation, appears evident from the circumstances already stated, but more especially from the fact just noticed, viz. adhesions being always found to accompany the latter form. It is, however, important to remark, that the formation of the cellulo-fibrous tissue which takes the place of the muscular tissue of the heart, does not originate in the effusion and organization of coagulable lymph. I have been able distinctly to trace it to the separation of fibrine from blood forced into the substance of the heart, in consequence of softening and rupture of some of its muscular fibres. The gradual transformation of the fibrine into the cellulo-fibrous tissue was very conspicuous.

The increase of thickness which takes place in dilatation of the veins affects the middle and external coats, and frequently equals and sometimes surpasses in this respect that of arteries of the same dimensions.

The vascular structure of some accidental tissues, although sometimes obviously a prolongation and enlargement of the minute arteries, veins, and capillaries of the affected

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part, is much more frequently, like the tissue of which it forms a part, a new formation.

The great enlargement of the spleen which succeeds to frequent attacks of intermittent fever, as well as other kinds of enlargement of this organ, does not depend so much on hypertrophy of the cellulo-fibrous, as on dilatation of the cellulo-vascular structure.

Hypertrophy of bone is often carried to a great extent, sometimes in chronic inflammation of the bone itself, frequently of the periosteum and other tissues external to it. It is rarely observed in cartilage.

An increase of bulk of the muscular tissue from irritation is seldom considerable, except in the involuntary muscles. Thus, in chronic bronchitis the longitudinal, but more especially the circular fibres of the bronchi often undergo a great increase of bulk; in chronic dysentery the muscular coat of the colon often presents from one to two or three lines in thickness; and in chronic cystitis, more particularly when produced by the presence of calculi long retained in the bladder, it has been found to measure from four to six or even eight lines in thickness.

The most obvious cases of hypertrophy of the heart from local irritation are those which follow pericarditis. The symptoms of hypertrophy manifest themselves after the cure of the pericarditis, and after death the pericardium is found united by firm cellular tissue to the heart. The size of the heart is generally increased, sometimes natural, and in two instances I have found it diminished. This last state of the heart was found in persons who died of phthisis; and in one case of tubercular pericarditis the hypertrophy was concentric.

That hypertrophy is produced in glandular organs from irritation is sometimes sufficiently obvious. Hypertrophy of the liver arises undoubtedly from this cause when its lobular structure is enlarged, the cellular tissue at the same time being more or less thickened, or the peritoneal surface of the organ united by adhesions to the diaphragm and abdominal parietes. Considerable enlargement of the salivary glands is not uncommon in scrofulous ulceration of the neck; and a still greater is well known to follow frequent attacks of inflammation of the amygdalæ. Enlargement of the pancreas from irritation or chronic inflammation is extremely rare, or rather has not perhaps been established as a pathological fact. Hypertrophy of the lymphatic glands is a frequent occurrence in the vicinity of ulcers; that of the kidneys and of the testicles is rare, and never considerable.

A number of examples of considerable increase of bulk of the brain, spinal cord, nerves, and ganglia, from the influence of morbid stimuli, are recorded. This change of bulk has been met with in portions of the brain, in one or both hemispheres; in a part or the whole of the cord, sometimes so considerable in degree as to have been followed by compression, apoplectic and epileptic symptoms, and death, the consequences of the resistance opposed by their osseous encasement to a further increase of their bulk. Enlargement of the ganglia from a similar cause has been much more seldom observed, whereas it is more common in the nerves, although very slight in degree, from their frequent proximity to ulcers or other chronic inflammatory affections.

Such are the various forms of hypertrophy which have been observed to occur in the different organs and tissues of the body, and the obvious and local causes under the influence of which they are produced. It must, however, be acknowledged that there are others in the production of which the operation of any of these causes cannot be satis-

[*Hypertrophy*, 2.]

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factorily established. Of this kind is the remarkable case of dilatation of the lymphatics, the particulars of which have been detailed. Such also are those cases of the accumulation of fat in the form of tumour under the skin, around the kidneys and basis of the heart, and in other parts of the body; the disproportionate increase of bulk of entire organs, portions, or regions of the body, apparently unconnected with original conformation; the anormal development of the hair of the head and pubis, the epidermis and nails. The *Cutis Pendula*, *Dermatolysis*, &c. of authors, is a striking example of hypertrophy of the skin, the cause of which is unknown. It is characterized not so much by an increase of thickness as by great extension of the skin, which is thrown into folds, sometimes several inches in breadth, which, when grouped together, form large pendulous masses: it occurs chiefly in the skin of the upper part of the face, chest, and abdomen.

Physical Characters of Hypertrophy.—The physical characters of hypertrophy are referable to changes taking place in the relative bulk, weight, consistence, colour, and form of the affected parts. I have already sufficiently indicated the relative *increase of bulk* which constitutes the essential physical character of this change. It is, however, important to observe, that hypertrophy may exist to a great extent without being accompanied by any increase in the size of the affected part,—a fact which is rendered most conspicuous in hollow muscular organs. Thus the walls of the heart may be two or three times thicker than in the natural state, the size of the heart itself remaining unaltered. The same is observed in hypertrophy of the muscular coat of the urinary bladder and colon, the hypertrophy in these cases having taken place from without inwards. Under these circumstances the capacity of the organ is diminished in proportion to the degree of the hypertrophy. But, besides, the existence of hypertrophy may be accompanied with even a diminution of bulk, sometimes to such an extent in the bladder and colon, in cases of chronic inflammation of their mucous membrane, that their respective cavities are almost effaced,—an effect, however, partly produced by the contraction of the muscular coat of these organs. Again, hypertrophy may exist without being accompanied by any change in the relative capacity, bulk, or external dimensions of an organ or tissue. This happens in muscular, cellular, and glandular organs and tissues particularly, and is indicated merely by an increase of their consistence, and a more compact organization. Lastly, hypertrophy of the muscular coat of hollow organs may be very great, although this coat may have preserved its natural thickness, and even when it is thinner than natural, when accompanied with dilatation. Hence the existence and degree of hypertrophy in these organs in particular is to be determined by a comparative estimate of the relative thickness of their walls and the dimensions of their cavities, or simply, of the superficial extent and thickness of their muscular coat.

The *weight* of an organ affected with hypertrophy must necessarily be increased, although it is a means seldom employed to determine a relative increase of bulk. Hypertrophy of the brain or portions of it, of the liver, kidneys, heart, and other hollow organs, would, however, be more accurately and more conveniently ascertained by weight than measurement. By these means, conjointly employed, we might always ascertain the precise extent of hypertrophy of an organ, the mean bulk of the same, under similar circumstances of age and stature, being given as a standard of comparison. But such a standard has not as yet been established, to enable us to do so otherwise than approximately. For, although in no organ is the occurrence of hypertrophy so frequent, and at the same time so dangerous as in the heart, and consequently in none is it so important to be able to ascertain the existence and extent of this lesion, yet the

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normal dimensions of this organ are differently stated by different pathologists. Thus Laennec considers that the size of the fist may be taken as a standard of comparison for determining the normal dimensions of the heart; Lobstein fixes its weight at nine or ten ounces; Cruveilhier at six or seven ounces; and Bouillaud at eight or nine ounces in persons between twenty-five and sixty years of age, one or two ounces less in persons between sixteen and twenty-five, and in tall and robust persons at ten or eleven ounces.

This discrepancy of opinion respecting the normal weight and dimensions of the heart considered as a whole, appears also in those which have been assigned to the thickness of the walls of its respective cavities. Laennec merely observes that the thickness of the walls of the left ventricle should be a little more than double that of the right. Lobstein gives the following measurements of the normal thickness of the walls of the ventricles and auricles:—right ventricle two lines and a quarter, left ventricle seven lines at its basis, and four lines a little above the apex; right auricle one line, left half a line. Bouillaud fixes the mean thickness of the right ventricle at two lines and a half, and that of the left at seven lines; of the right auricle at one line, and that of the left half a line.

The *consistence* of a part in a state of hypertrophy may be natural, increased, or diminished. An increase of consistence is by far the most frequent, and is sometimes very considerable in hypertrophy of the cellular tissue, lymphatic glands, and substance of the brain; and the bones sometimes acquire a degree of hardness equal to that of ivory. A diminution of consistence is of rare occurrence, and cannot be regarded as a necessary consequence of hypertrophy.

The *colour* of hypertrophied parts is sometimes increased, and this probably occurs when the bloodvessels are not compressed. A diminution of colour is more common, and in soft parts, such as the brain, is obviously accompanied by compression of the bloodvessels which ramify in the hypertrophied substance of this organ. Such also is frequently observed in hypertrophy of bone.

A change of *form* must necessarily accompany hypertrophy in every case in which the latter is partial or circumscribed: hence the production of osseous tumours or nodes projecting from the bones, and numerous deformities of the shafts and extremities of the long bones, the processes and bodies of the vertebræ, &c. The skin, in parts naturally smooth, forms numerous plicæ, or tumours of various sizes, which, however, more frequently depend on circumscribed hypertrophy of the subcutaneous cellular tissue. A similar modification of form is also met with in hypertrophy of the mucous membrane and its subcellular tissue in the digestive canal, giving rise to the development of tumours varying from the size of a pea to that of a cherry or walnut. I possess an interesting example of this circumscribed development of the mucous and submucous tissue in the form of a tumour, having the cauliflower arrangement, in the stomach of a cow, obviously produced by irritation kept up by a pin, which is so fixed that the point must have been brought in frequent contact with the mucous membrane. The point of the pin corresponds exactly with the pediculated attachment of the tumour.

Some of the most marked changes of form accompanying hypertrophy with dilatation are observed in hollow organs, several varieties of which occurring in the bronchi, require particular notice, inasmuch as, if not carefully examined, they might be confounded with some other diseases of the lungs, more especially with vesicular emphysema in some situations, and with vomicæ in others. In the first variety of form which accompanies dilatation, the bronchi of the second, third, or fourth order, present

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a fusiform shape, each bronchus gradually increasing in size in the direction of its distribution, until it has acquired very considerable dimensions, varying from a quarter of an inch to an inch in diameter. The second variety, which occurs in very small bronchi, and chiefly in those which pass out from the sides of the large bronchi, presents a pyriform shape, the dilated tube having a narrow pediculated attachment, and a rounded fundus from three to six lines in diameter. In the third variety, which is met with generally in bronchi of the fourth order, the dilatation is globular, the whole circumference of these tubes being equally dilated within a circumscribed extent. Sometimes one, but more frequently several globular dilatations, either contiguous to, or separated from each other by irregular intervals, occupy the same bronchus, and vary from a few lines to nearly two inches in diameter. The last variety occurs in the smallest bronchial tubes, several of which being dilated in a circumscribed portion of lung, small groups of round or ovoid bodies, of the size of a hemp-seed, pea, or cherry, are formed, which, when carefully separated from the surrounding pulmonary tissue, present the appearance of a bunch of grapes. This variety is sometimes complicated with emphysema, the dilated bronchi being filled with a thick mucous or muco-purulent secretion, and the air-cells with air, and when not very considerable, may be confounded with the latter disease. It may also, when situated in the upper lobe of the lung, be confounded with a multilocular tubercular excavation, when lined by a smooth mucous membrane of new formation. The globular variety likewise, when it occupies the same situation, is still more likely to give rise to a similar mistake.

The changes of form which accompany dilatation of the arteries and veins are equally conspicuous with those which are observed in the bronchi, but do not require special notice. It may, however, be remarked, that the lateral dilatation of the former exercises an obvious influence on the blood, in consequence of this fluid being retained, and thereby favouring its coagulation and the formation of those fibrinous layers which this kind of dilatation is found to contain.

The only other organs in which a change of form becomes conspicuous in hypertrophy are the brain and heart, in neither of which, considered apart from the hypertrophy which gives rise to it, is it of much importance.

DESCRIPTION OF THE PLATES.

PLATE I.

Figs. 1 and 2 represent hypertrophy and dilatation of the bronchi. *Fig. 1*, a section of one of the lobes of the lung with the dilated bronchi laid open. The mucous membrane is congested, and the circular fibres so much enlarged as to project considerably above the surface in the form of irregular bands. Some of the dilated bronchi, A, A, A, are cylindrical; others, B, fusiform; a few, C, C, sacculated; or, D, D, pyriform. The two latter forms of dilatation occur in the small bronchi which open into the larger ones. E, muco-puriform matter filling dilated bronchi. *Fig. 2*, bronchial tubes separated from the substance of the lung and laid open; A, B, C, D, five bronchial tubes greatly dilated; the circular muscular fibres remarkably developed, forming numerous broad bands, intersected by deep sulci or lateral dilatations of the bronchi. The mucous membrane is much thickened in general, and at E is covered by a multitude of round or oblong projections, analogous to the villi of the digestive mucous membrane. The following *figs.* have been introduced into the present fasciculus from want of room in the following one, to which they properly belong. The first of them, *fig. 3*, represents that form of dilatation of the bronchi which is accompanied with atrophy. The dilatation occurs at the extremity of a bronchial tube, has a globular form, all the tissues of which it is composed are so thin that, when laid open, the colour and structure of the pulmonary tissue can be seen through them. They are generally found filled with a thick, tenacious, straw-coloured, muco-purulent fluid. The portion of lung represented in this *fig.* has been divided longitudinally. A, B, the corresponding halves of a dilated bronchus; C, D, a very large dilatation cut in two; E, the muco-puriform matter occupying one-half the dilated bronchus F; the same kind of matter, G, G, filling smaller dilatations. *Figs. 4, 5, 6, 7*, represent the more remarkable forms of emphysema. *Fig. 4*, emphysema of the upper lobe of the right lung; A, B, and C, represent the progressive dilatation of the air-cells. At A, the dilatation of the cells occupies several lobules, and varies from the size of a pin's head to that of hemp-seed; at B, and at D, where they are exposed by a transverse section, they are as large as a pea; and at C, several of them are as large as a cherry or walnut. This extraordinary size is, however, the consequence of rupture of the walls of two or more of the dilated cells.

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The pulmonary tissue, as happens in cases of this kind, contains a great quantity of black pulmonary matter. *Fig. 5* is a remarkable example of emphysema occupying all the lobes of both lungs from an infant. In some parts, A, B, C, the dilated cells are congregated into groups, varying from the size of a pin's head to that of hemp-seed; in others, and principally at or near the margin of the lobes, some of them are as large as a garden-pea. *Fig. 6* represents the lobular emphysema which not unfrequently accompanies tubercular phthisis. A, A, miliary tubercles; B, B, the emphysematous lobules. *Fig. 7*, a section of a small portion of dried lung affected with interlobular emphysema. A, A, the effused air contained in the interlobular cellular tissue, and carrying the pleura outwards in the form of irregular ridges; B, B, sections of the interlobular cellular tissue.

PLATE II.

Fig. 1 represents a transverse section of the heart, both ventricles of which are greatly hypertrophied, and the cavity on the left side dilated. A, apex of the heart; B, cavity of the right, C, of the left ventricle; D, walls of the right, E, of the left ventricle; F, septum; G, G, columnæ carneæ greatly enlarged. *Fig. 2*, hypertrophy of the left ventricle without dilatation; and dilatation of right ventricle. A, anterior surface of the basis of the heart; B, cavity of the right, C, of the left ventricle; D, walls of the right, E, of the left ventricle; F, septum; the columnæ carneæ are not enlarged. *Fig. 3*, extreme concentric hypertrophy of the right ventricle, from congenital contraction of the orifice of the pulmonary artery. A, anterior surface of the heart towards the apex; B, cavity of the right, C, of the left ventricle; D, walls of the right, E, of the left ventricle; F, septum; G, one of the columnæ carneæ greatly enlarged. *Fig. 4* represents the pulmonary artery laid open to expose the malformation of the semilunar valves, the union of which has caused the contraction of the orifice of this vessel. A, pulmonary artery separated from the heart; B, the artery laid open; C, a projecting circular border, formed by the union of the semilunar valves; D, the small opening left for the passage of the blood. *Fig. 5*, hypertrophy of the heart and ossification of the pulmonary artery. A, surface, B, cavity, D, D, walls of right ventricle affected with hypertrophy to a great extent; C, E, cavity and walls of left ventricle; F, arch of aorta; G, G, trunk of the pulmonary artery; H, right branch of the same, much dilated; K, numerous points of ossification. *Fig. 6* represents the case of congenital concentric hypertrophy of the heart which occurred in a woman above forty years of age. A, right ventricle; B, right auricle; C, left ventricle; D, left auricle; E, aorta, F, pulmonary artery; G, coronary arteries very large. *Fig. 7*, transverse section of the heart; A, anterior surface; B, cavity of the right, C, of the left ventricle; D, walls of the right, E, of the left ventricle; F, septum; all of which are greatly hypertrophied; the cavities of the ventricles diminished in proportion, and the entire bulk of the heart at least one-half less than natural.

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PLATE III.

Figs. 1, 2, 3 of this plate represent the morbid appearances which precede and accompany the formation of aneurismal dilatation of the heart. *Fig. 1* is an interesting example of circumscribed aneurism occurring in two separate portions of the left ventricle. The case has been already published by Mons. Reynaud in the *Journal Hebdomadaire*, illustrated by a lithograph from the original drawing. A, A, A, walls of the left ventricle; B, apex of the same; C, a portion of the left auricle; D, mitral valve; E, cavity of the aneurism exposed by a longitudinal section of its sac and the walls of the ventricle in which it is imbedded; F, orifice of the aneurism communicating with the cavity of the ventricle, as indicated by the introduction of a bent piece of wire; G, one half of the aneurismal sac separated from the corresponding walls of the ventricle, and containing a small fibrinous coagulum. H, H, the morbid appearance, viz. the opacity and pale-straw colour, of the serous membrane; the thickening of the cellular tissue beneath it is seen along the cut margin of the aneurism, the walls of which it forms, still covered, however, by the serous membrane. K, one of the columnæ carneæ atrophied. M, a smaller aneurism laid open by a longitudinal incision; it also contains a small fibrinous coagulum, N, and its walls are formed by the serous membrane and cellular tissue, which present the same morbid appearances, P, as those seen in the situation of the larger aneurism. *Fig. 2* represents four aneurisms of the same kind as the former, situated also in the left ventricle. A, aorta and semilunar valves; B, walls of the ventricle; C, columnæ carneæ; D, pericardium united to the heart by firm cellular tissue; E, the cellulo-fibrous tissue which is formed from the fibrine of the blood, and towards this part the cavity of the ventricle is sensibly dilated. F, G, H, and K are circumscribed aneurisms, similar in every respect as regards the morbid state of the serous membrane and cellular tissue around them, to those represented in *Fig. 1*. Some of them are deeper than others, but none of them contained fibrinous coagula. *Fig. 3* represents the ventricles of the heart laid open by a transverse section; A, A, left, B, B, right ventricle; C, C, upper, D, D, lower section of the walls of the left ventricle; E, E, E, the pericardium intimately united to the heart by firm cellular tissue; F, F, columnæ carneæ, the lining membrane of which and the subcellular tissue present the same pale yellow colour, opacity, and loss of elasticity as in the other cases. The greater part of the walls of the left ventricle was replaced by a tissue, in some parts of a fibrinous, in others of a cellulo-fibrous or fibrous character, thus indicating the gradual transformation of the fibrine of the blood into fibrous tissue. The columnæ carneæ are flattened, and the cavity of the ventricle is greatly dilated, particularly in the direction of the walls where they are most diseased. *Fig. 4*, a section of a portion of the tibia with a circumscribed bony enlargement, or node, from a person affected with syphilis. The structure of the node is not so regular as that of the bone, but it is equally compact. Traces of the external wall of the tibia are still observable, shewing that the new osseous deposit took place on the inner surface of the periosteum. *Fig. 5* represents hyperostosis of the tibia occurring as an acute affection accompanied with much pain. A, a portion of the head of the bone; B, cancellated structure; C, medullary canal; D, thickened walls of the bone; E, medullary membrane, and F, portions of the bone, red and vascular.

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PLATE IV.

This plate represents aneurismal and varicose dilatation of the arteries; dilatation of the lymphatics and urethra. *Fig. 1*, aneurism of the basilar artery. A, cavity of the aneurism; B, portion of basilar artery; C, the vertebral arteries. The only morbid appearances in the dilated walls of the artery were those which I have described. *Fig. 2* represents a portion of the brachial, ulnar, and radial arteries extensively affected with aneurismal dilatation, taken from a case published by Jules Cloquet in his *Pathologie Chirurgicale*. All the arteries of the body were similarly affected, but those of the extremities most extensively. The aneurismal tumours varied from the size of a hemp-seed to that of a large pea, and their walls were thinner than those of the unaffected portions of the artery. A, brachial artery laid open; B, ulnar, C, radial artery; D, D, D, D, aneurismal tumours projecting from the external surface of the preceding arteries; E, E, E, aneurismal sacs communicating with the brachial artery. *Fig. 3*, varicose dilatation of the iliac arteries taken from the work just referred to. The common iliac arteries and their two branches, the internal and external iliacs, present a remarkable increase of bulk, and are flexuous and elongated, forming numerous sinuses and dilatations of various dimensions and forms. The walls of the dilated portions were soft, flaccid, and collapsed; their colour was paler than natural, the middle coat had lost its yellow tint, and its circular fibres were less apparent; in some points it resembled a thin plate of fibro-cartilage, being very elastic in the direction of its thickness. A, termination of the abdominal aorta; B, inferior mesenteric artery; C, left common, D, right common iliac arteries; E, left external, F, left internal iliac arteries; G, right external, H, right internal iliac arteries; K, K, K, K, sinuosities separating the dilatations M, M, M, M, of the preceding arteries. *Fig. 4* represents a portion of the thoracic duct and a small group of the lymphatics in the remarkable case of dilatation of these vessels described in the present fasciculus. A, a portion of the diaphragm; B, thoracic duct, and C, C, two large branches; D, D, some of the lymphatics, a number of which are besides given in outline to convey a more comprehensive idea of the extent to which these vessels were dilated. *Fig. 5*, dilatation of the urethra. A, glans penis; B, body of the penis; C, bulb of the urethra; D, D, the two crura of the penis; E, E, the walls of the dilatation, formed superiorly by the corpus spongiosum, laterally and inferiorly by the skin and cellular tissue; F, F, cavity of the dilatation; G, G, the extent of the urethra, communicating with the cavity E; the probe H is introduced to render the direction of the urethra more apparent. This cavity contained the calculus, *fig. 6*; and although the sound had been frequently introduced, its presence had never been detected, probably for the following reasons, viz. the situation of the dilatation, which occupied the inferior surface of the penis, and the stone not projecting into the urethra. The patient was treated for *sarcocoele*, in the Hôtel Dieu of Paris.

Plate I.



Fig. 1.



Fig. 2.

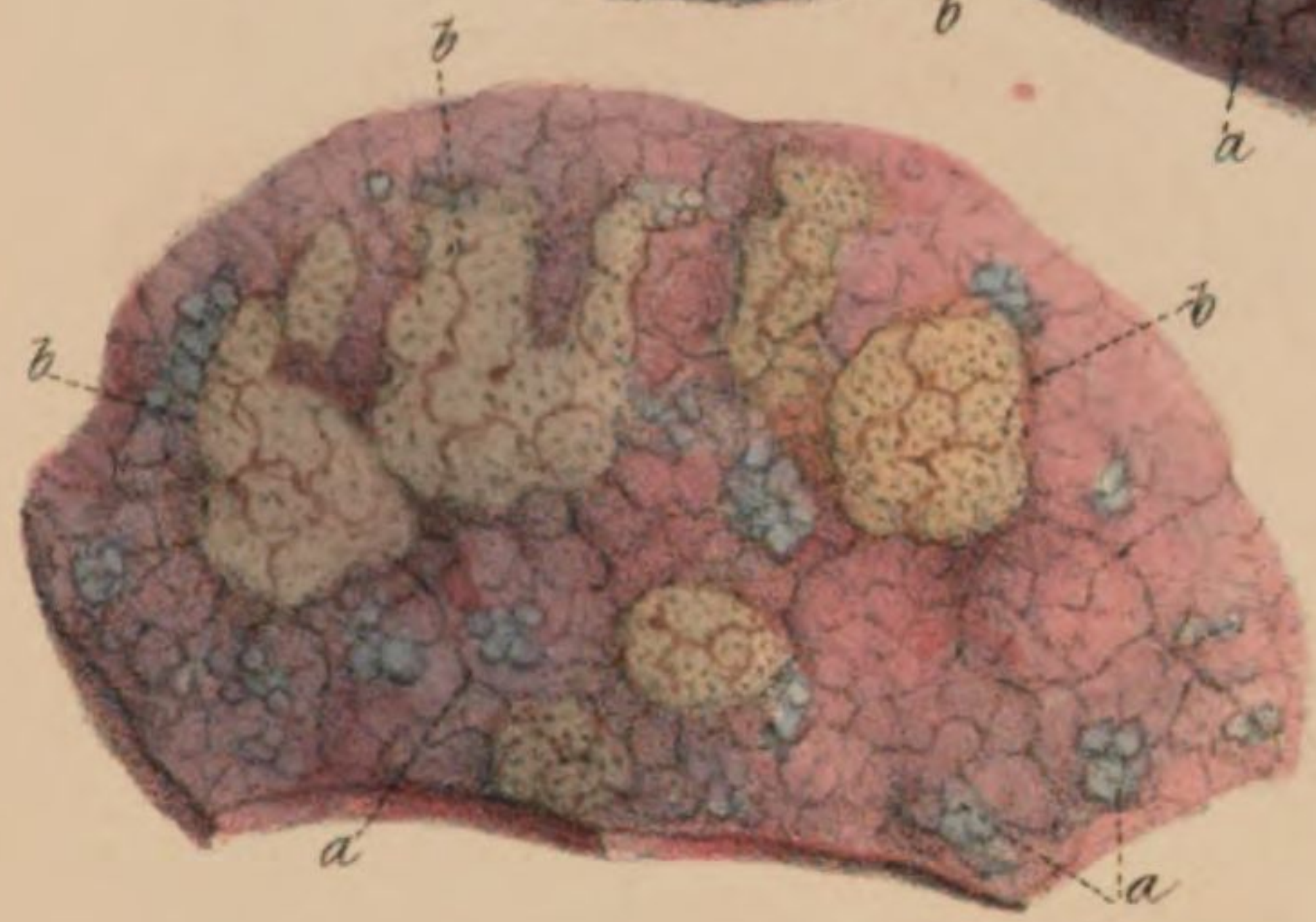


Fig. 6.

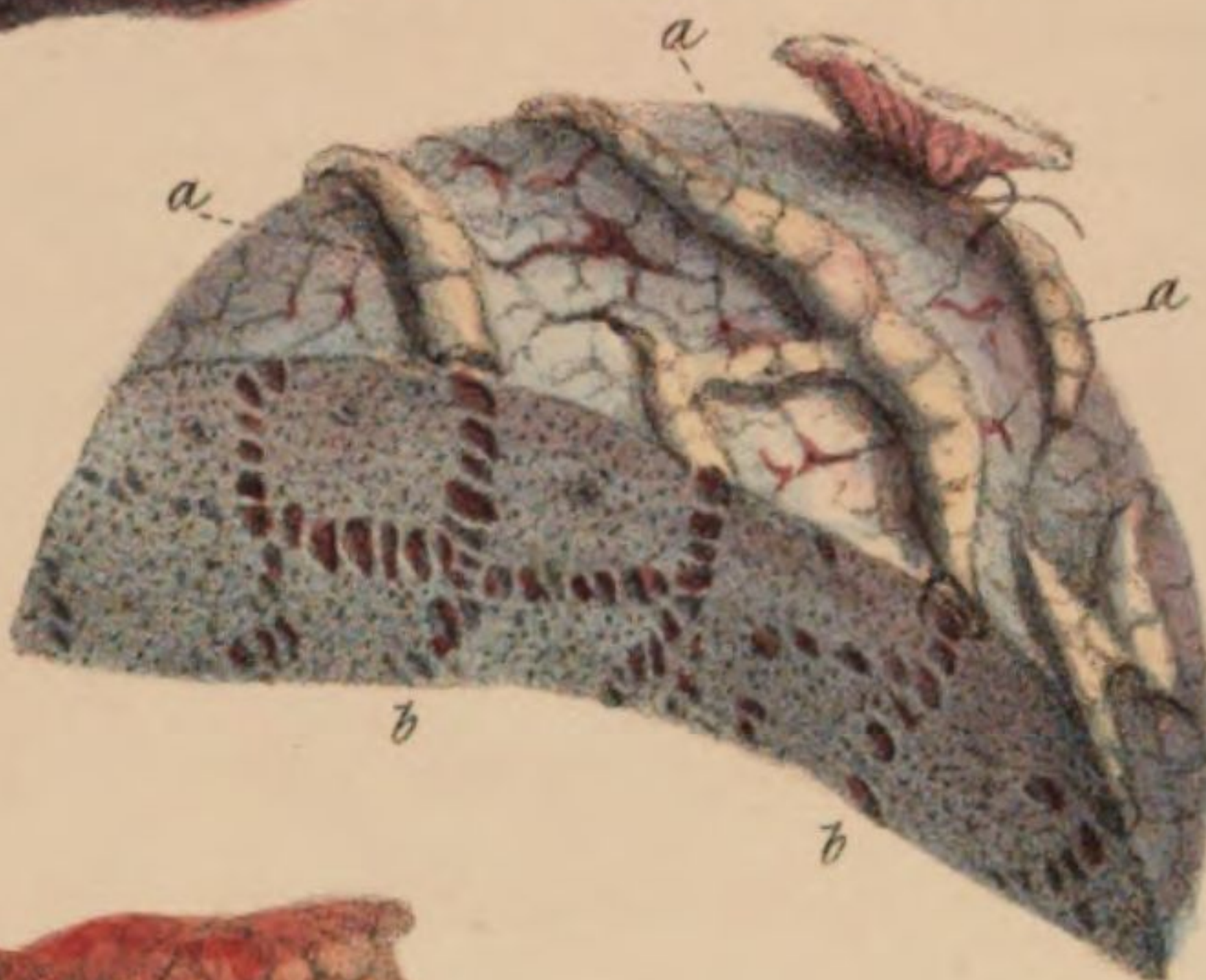


Fig. 5.

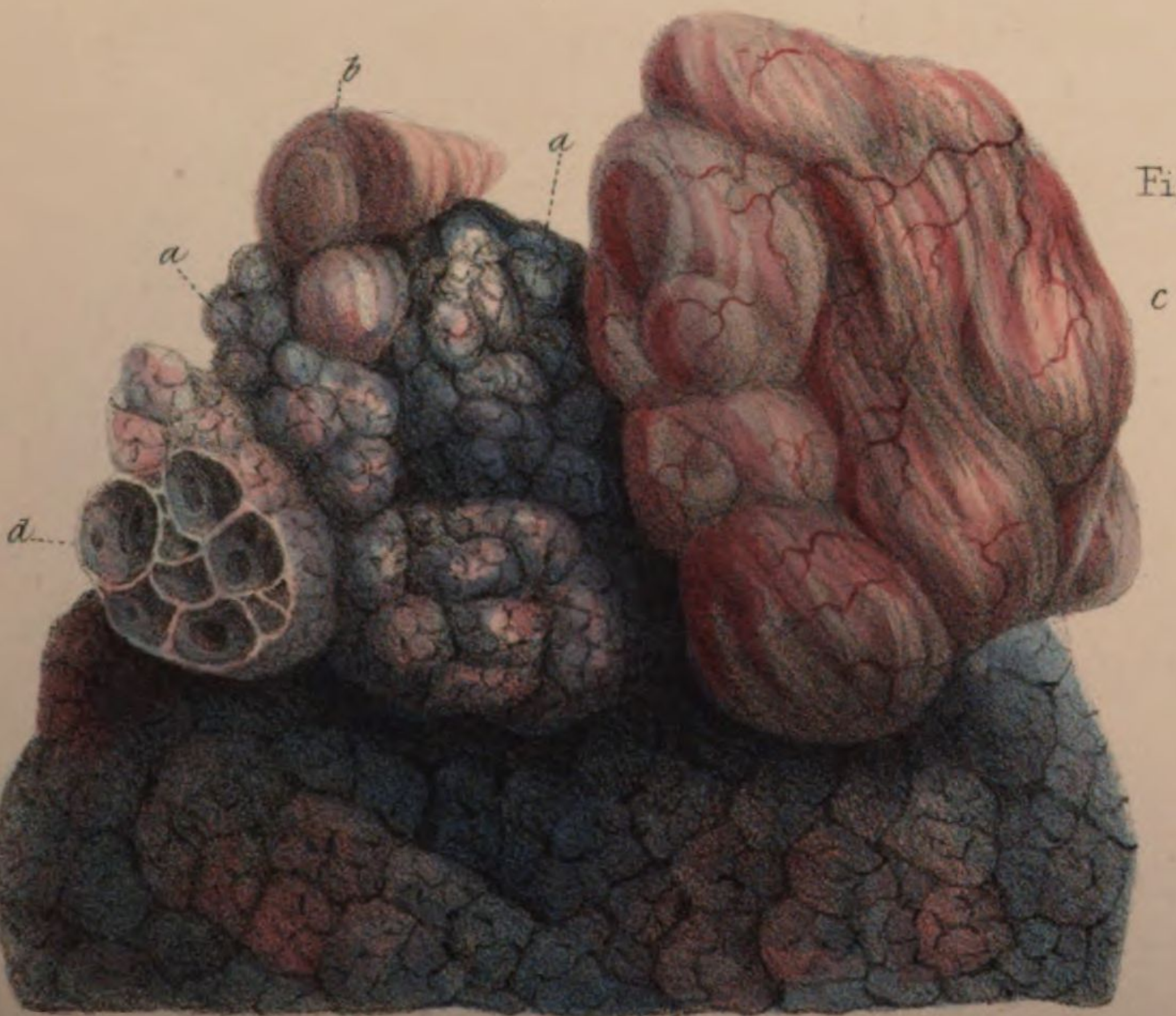
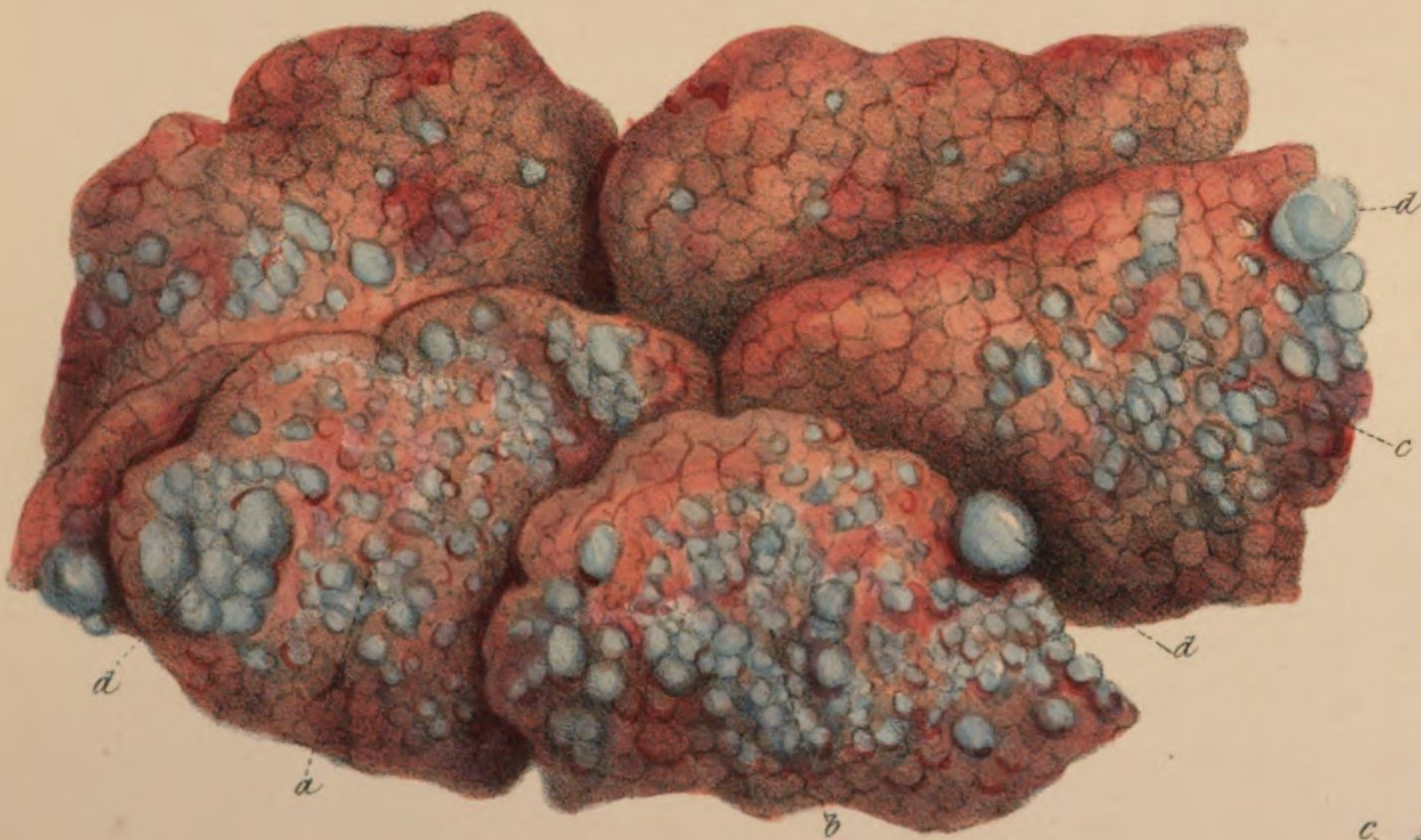


Fig. 4.



Fig. 3.

Plate II.

Fig 1.

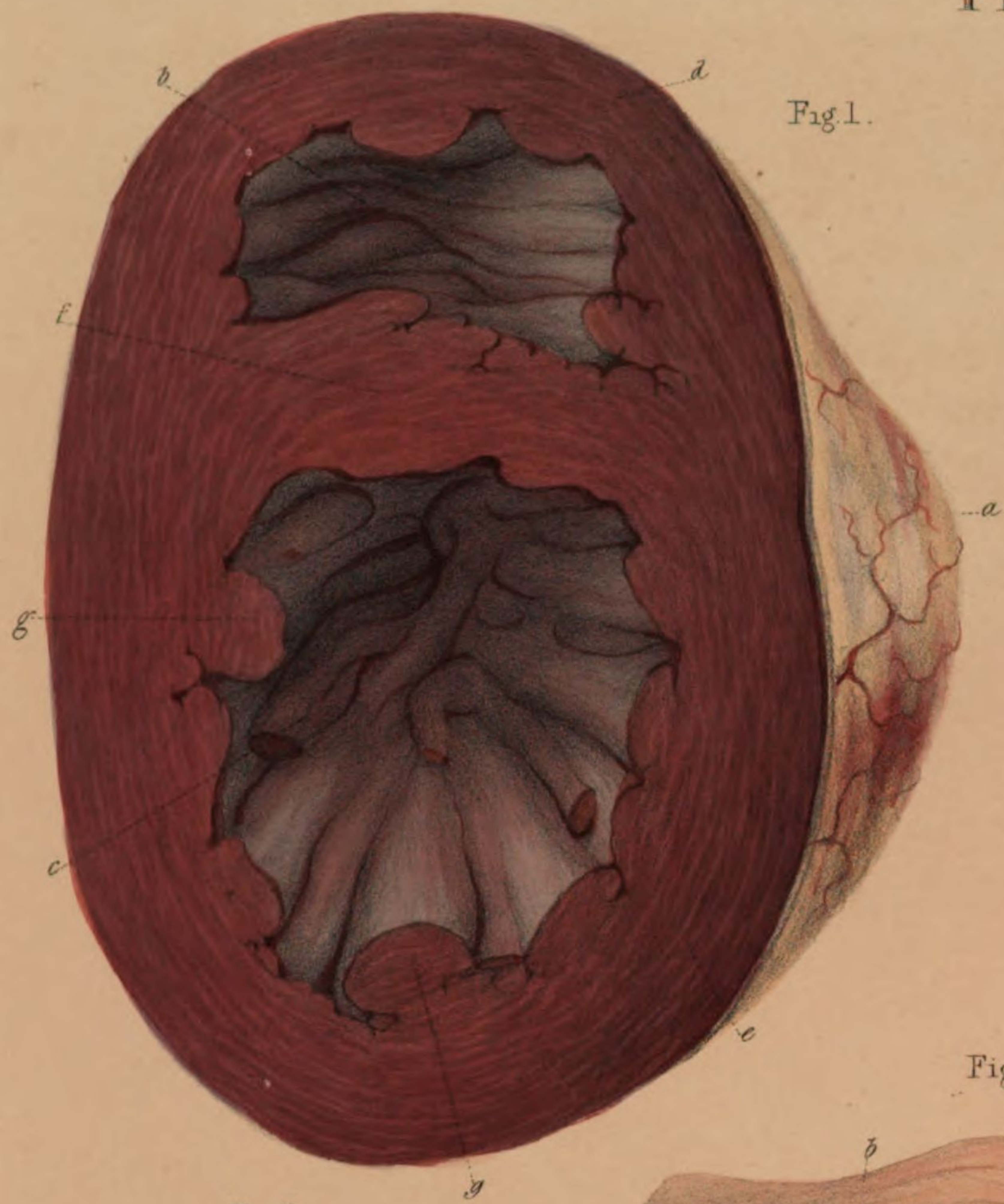


Fig 3.



Fig 2.



Fig 4.

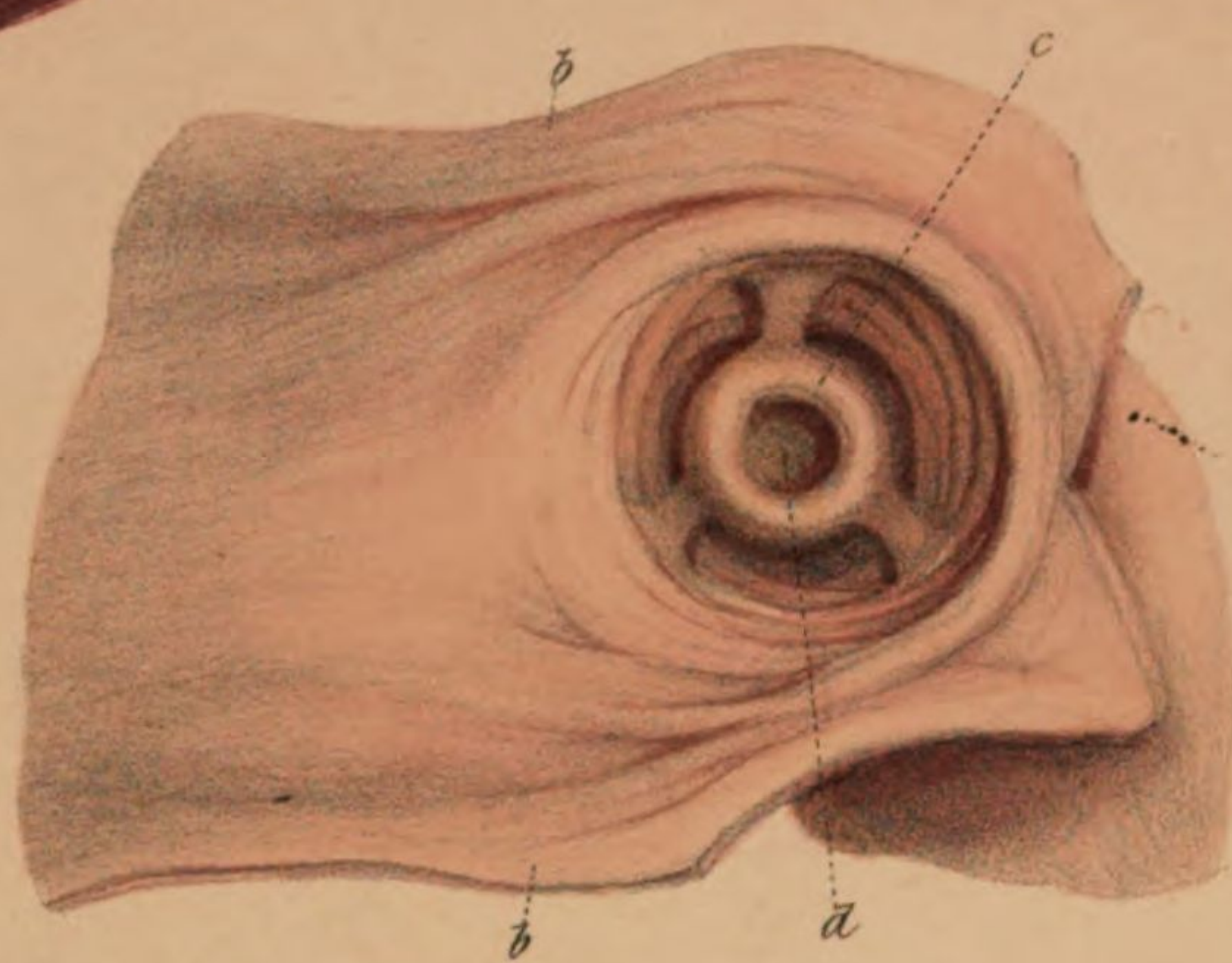


Fig 7.



Fig 5.

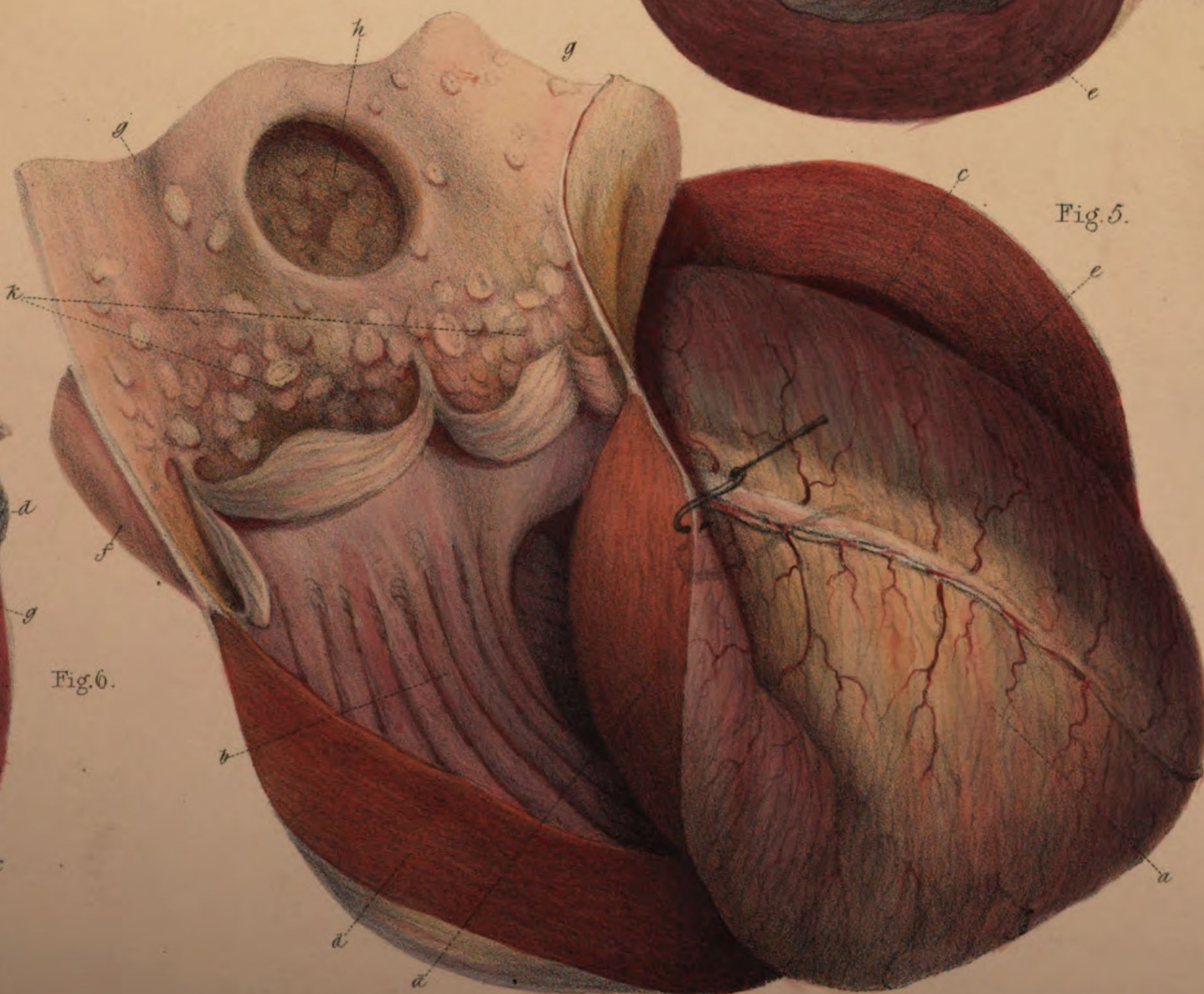


Fig 6.

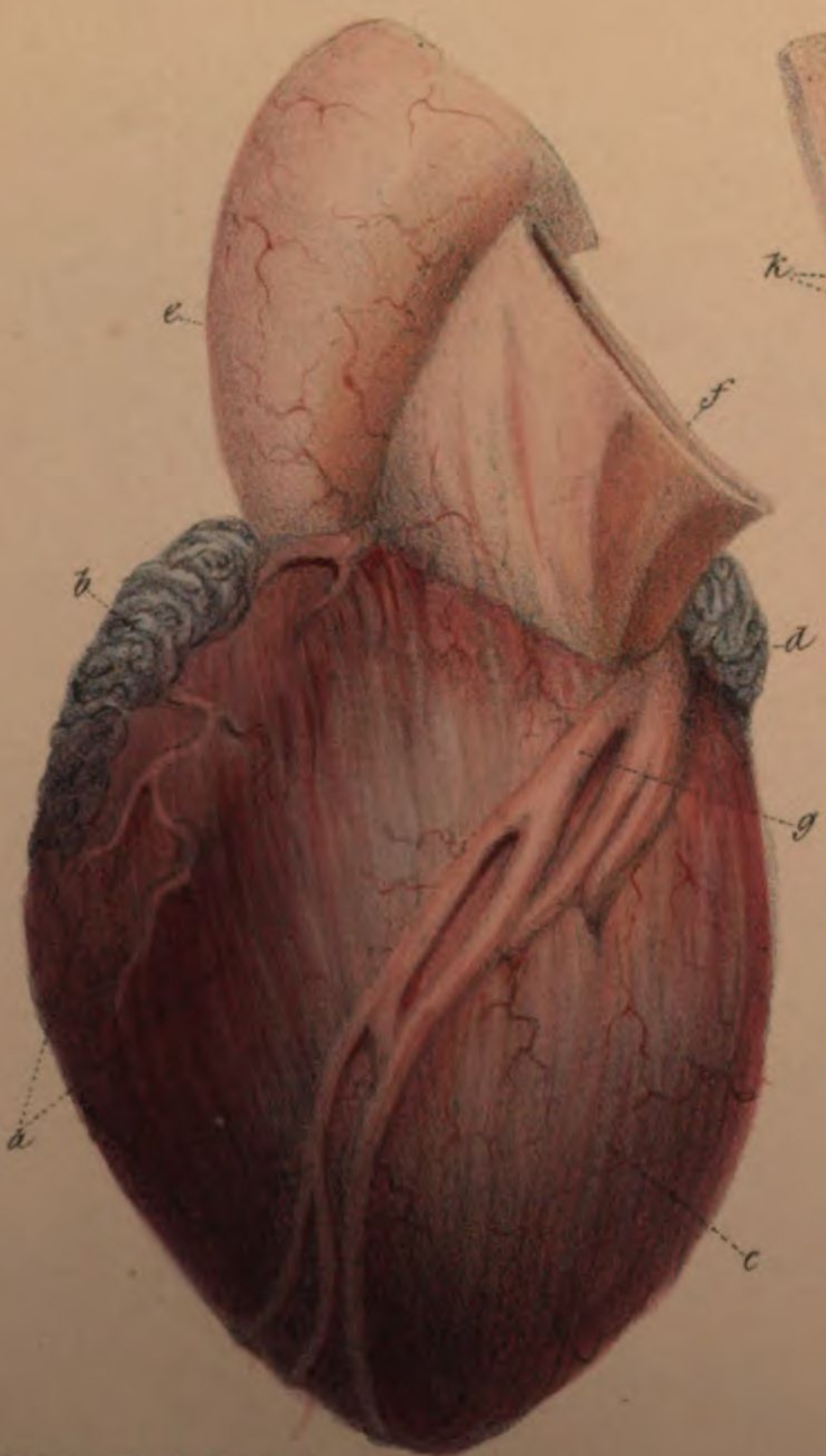


Plate III.



Fig. 5.

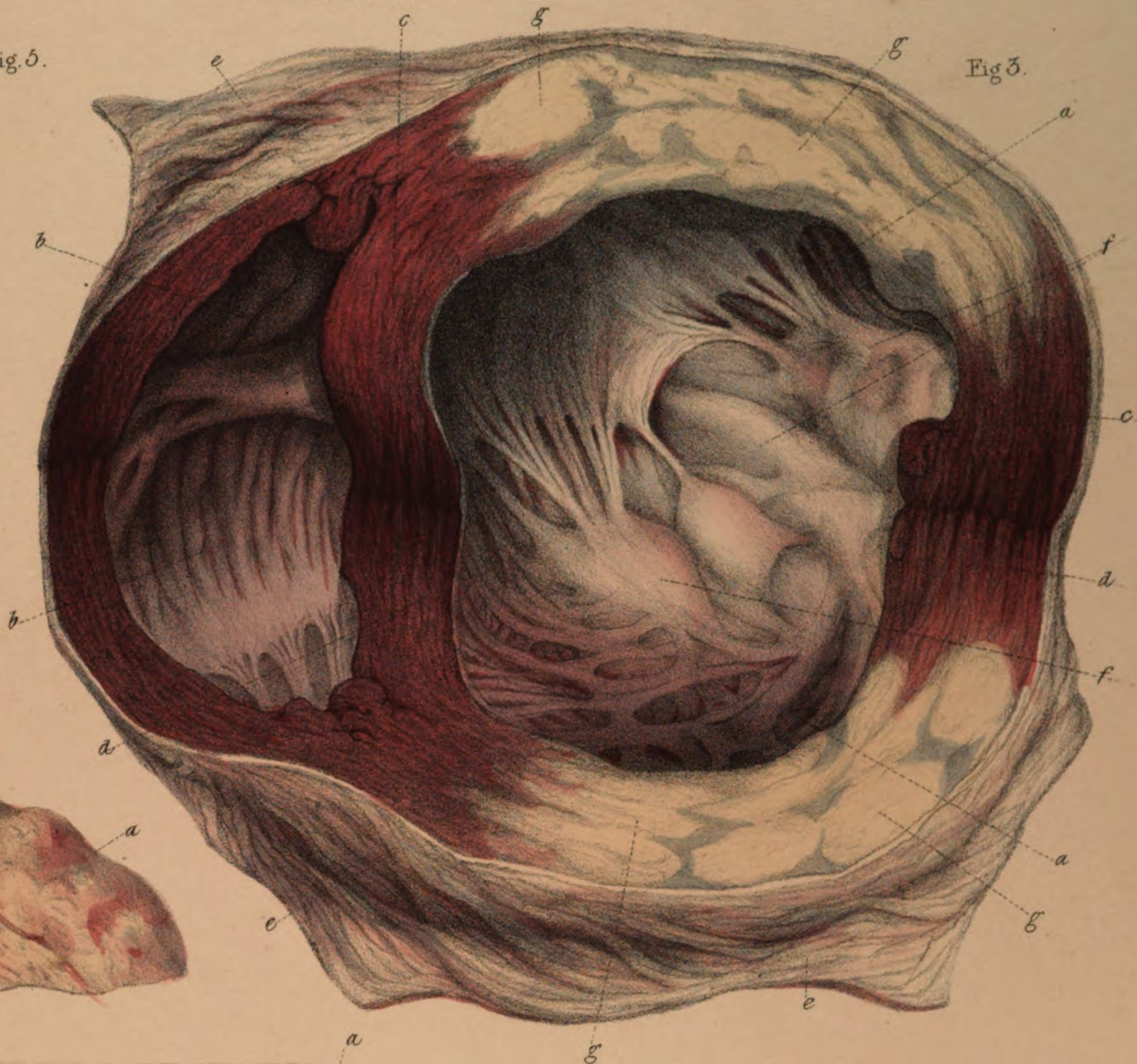


Fig. 3.



Fig. 4.

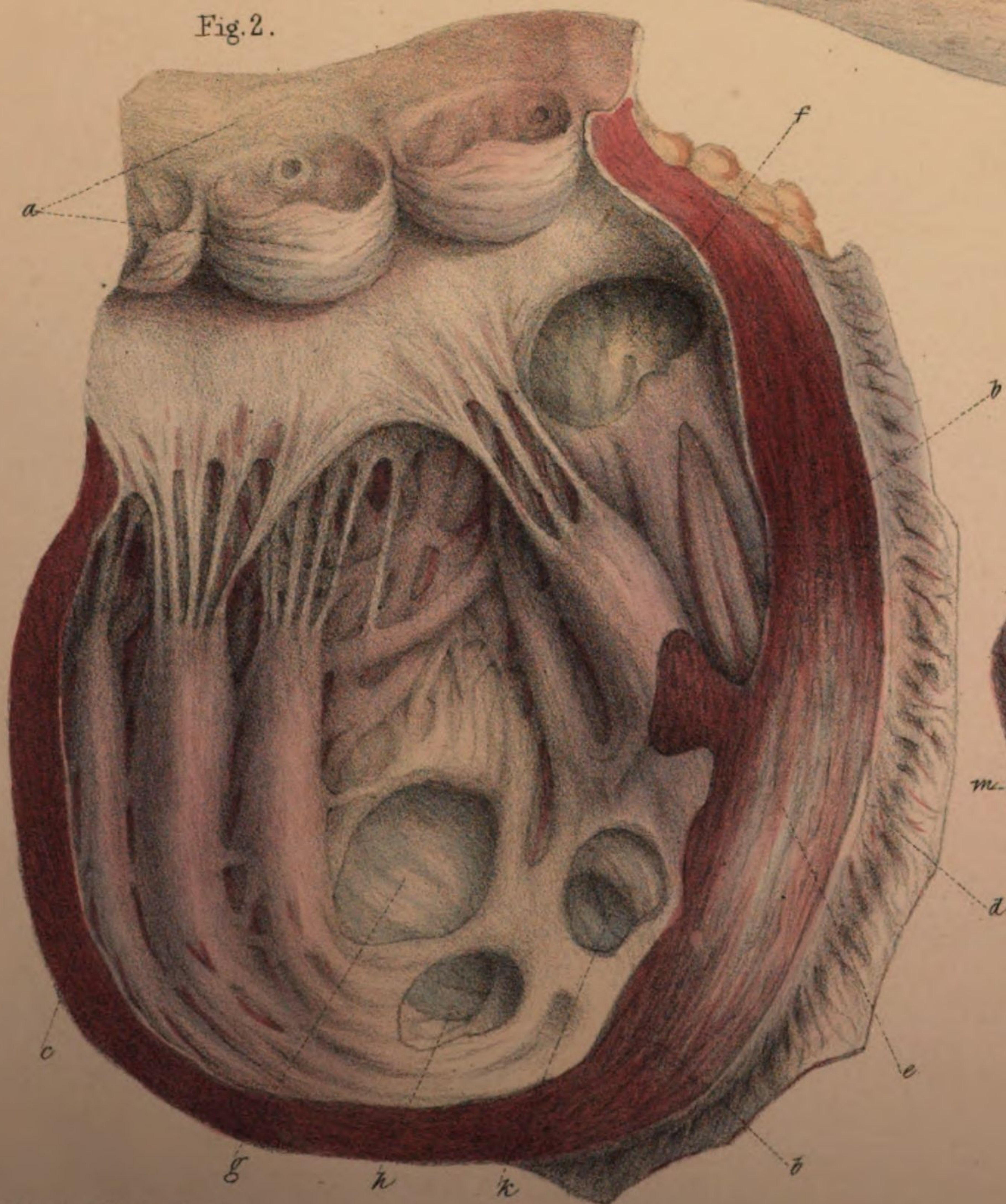


Fig. 2.

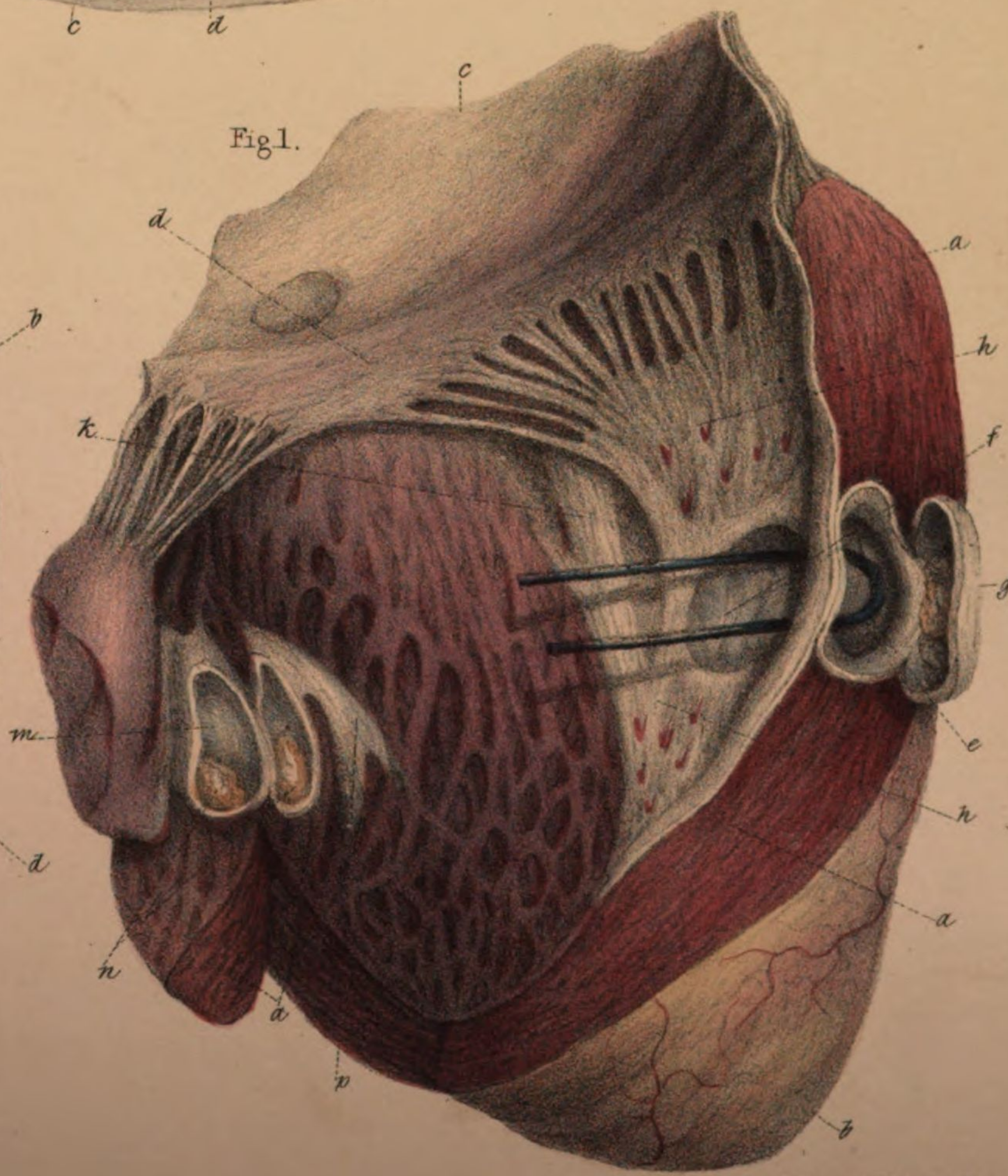
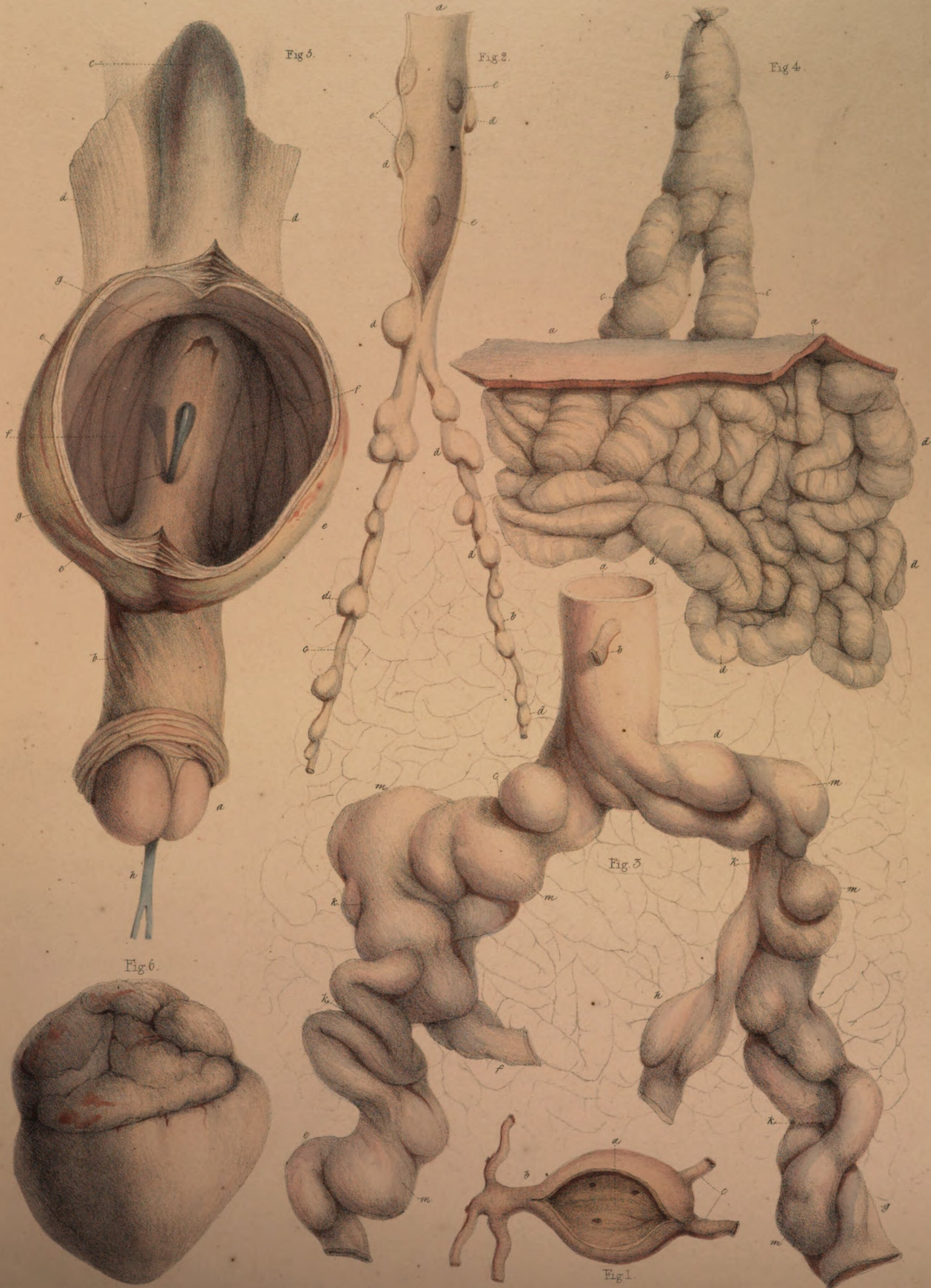
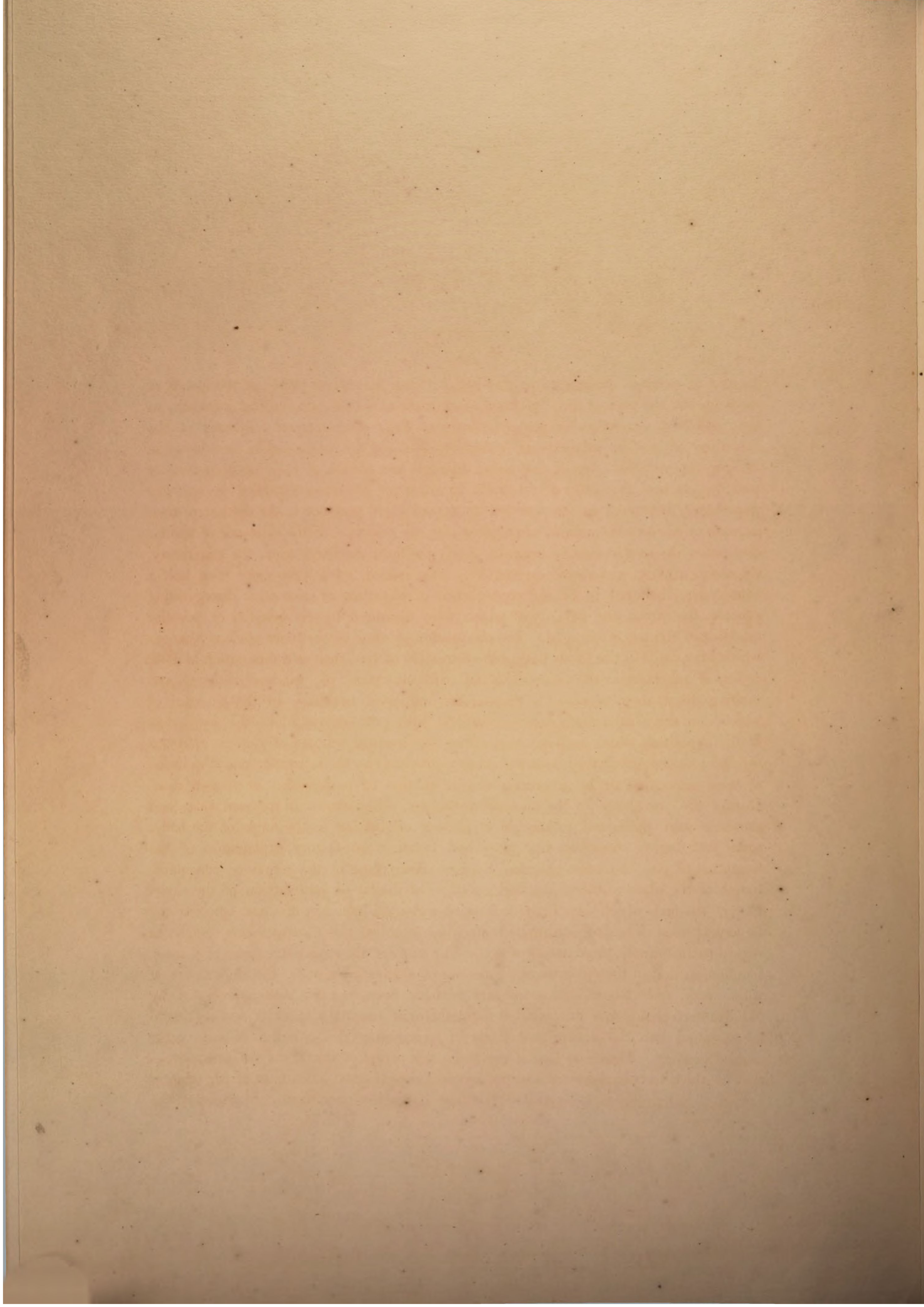


Fig. 1.

Plate IV.





P U S.

THERE is, perhaps, no morbid product which, from the earliest period of the history of medicine till the present day, has been so often the subject of pathological research, as that which is known under the appellation of *Pus*. The extreme frequency of the formation of pus in solutions of continuity followed by inflammation or ulceration affecting the external parts of the body, afforded the means of an early acquaintance with the physical characters of this fluid, to which the physician attached no ordinary importance, inasmuch as he was led to regard their presence in the discharges from mucous canals or preternatural communications, as evidence of the existence of similar destructive diseases in internal organs. Every puriform discharge from the respiratory, digestive, urinary, generative organs, &c., was, indeed, until little more than half a century ago, believed to be the consequence of ulceration or some other disorganizing process, the extent and severity of which were measured by the quantity or peculiar qualities of the pus discharged. The observation of some of the more obvious changes which take place in the solids during the formation of pus, such as a diminution of bulk or loss of substance in the tissues affected with ulceration, the gradual softening and melting down, as it were, of circumscribed indurated swellings, in the situation of which were found afterwards excavations filled with pus, naturally laid the foundation of the hypothesis which so long reigned in the medical schools of Europe, that this morbid product was formed from the solids of the body by the separation and dissolution of their molecules, or by a process similar to that of putrefaction or fermentation. During the prevalence of the humoral pathology, when almost all diseased states and products were attributed to morbid conditions originating in the fluids of the body, some pathologists conceived that they had found a satisfactory explanation of the formation of pus, in certain chemical changes taking place in the serum or coagulable lymph of the blood; others, in a melting down of the fat or putrefaction of the chyle. Few of the facts which were brought forward under the influence of these opinions can be looked upon as having contributed much to establish the doctrines now taught in regard to the formation and nature of pus. The study of the elementary forms of disease, founded on that of the structure and functions of elementary tissues, the importance of which may, with strict justice, be said to have been first felt and appreciated by Mr. Hunter, could alone have guided the pathologist towards a scientific and successful investigation into the nature and mode of formation of this as well as of every other morbid product. Hence we find it was about this period of the history of medicine,—that in which the physiological and pathological experimental researches of Mr. Hunter spread so much light on several of the most important phenomena of life,—that the

[*Pus*, 1.]

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formation of pus was shewn to consist essentially in a vital process similar to that of secretion

The opinion, however, that pus is formed in the capillary vessels by a process analogous to secretion, was first suggested by Dr. Simpson, of St. Andrews, in the year 1722. "If any foreign body," he observes, "be introduced between the edges of a wound, and the external air excluded, pus will continue to be discharged as long as you please; so that by this means, a *kind of new gland* is, as it were, produced. But if the wound be irritated or too much compressed, the properties of the liquor discharged will be immediately altered, as is well known to surgeons. Hence it follows that nothing is more easy than to change the secretions and humours of the body without the addition of any new matter or ferment, merely by changing the diameters and number of the secreting vessels." De Haen announced a similar opinion about the year 1756, from having observed that pus is often expectorated for a great length of time, by patients affected with phthisis, in whom, after death, no mark of ulceration could be perceived, not even the place in which the pus had been formed. From these circumstances, De Haen was led to believe that pus must have been immediately secreted from the blood, and adds "that although the blood appears to be a homogeneous fluid, yet it is manifest that there is something in it when collected which is of a tenacious consistence, of a whitish, or a yellowish colour, and which might be called the matter of pus." The opinions contained in the writings of De Haen seem to have suggested to Dr. Morgan the new theory of the formation of pus, which, as has been remarked by Professor John Thomson, was first embodied into a general doctrine, and subjected to a full discussion in his inaugural dissertation printed at Edinburgh in the year 1763, entitled, *Puopoeses, sive Tentamen Medicum de Puris Confectione*. The principles of the new theory of the formation of pus which had been thus introduced, soon found several able supporters, among whom Mr. Hunter may be regarded as the first who furnished satisfactory evidence of their accuracy, by means of new facts and illustrations derived from his experiments on living animals, and his researches on suppurative inflammation and on pus.

Before entering upon the details which belong to the present subject, it may be of some advantage to give a succinct view of the opinions entertained by Mr. Hunter on the properties of pus and the nature of the process by which it is formed, as we shall thereby be better prepared to appreciate the value not only of his labours but also of those which have been undertaken by others since his time.

Although Mr. Hunter, in his *Treatise on the Blood, Inflammation, &c.* has rather inconsistently described what he calls "Collections of Matter without Inflammation," we find him a strenuous advocate for the theory that inflammation always precedes the formation of pus, or that this morbid product is a consequence of what he called the suppurative inflammation; for he endeavours to establish, as an invariable fact, "that no suppuration takes place which is not preceded by inflammation; that is, no pus is formed but in consequence of it; that it is an effect of inflammation only, is proved in abscesses, from a breach in the solids attended with exposure, and from extraneous matter of all kinds, whether introduced or formed there. In abscesses, suppuration is an immediate consequence of inflammation; from the exposure of internal cavities no suppuration comes on till inflammation has formed the disposition and action; and although we find collections of extraneous matter, something like pus, in different parts of the body, yet such extraneous matter is not pus." In the chapter on Pus, Mr.

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Hunter observes, " that the cellular membrane, or circumscribed cavities, have their vessels but little changed from the adhesive state at the commencement of the suppurative disposition ; so that they still retain much of the form they had acquired by the first state, the discharge being at the beginning little more than coagulable lymph mixed with some serum. This is scarcely different from the adhesive stage of inflammation ; but as the inflammatory disposition subsides, the new disposition is every instant of time altering those vessels to their suppurative state ; the discharge is also varying and changing from a species of extravasation to a new formed matter peculiar to suppuration ; this matter is a remove further from the nature of the blood, and becomes more and more of the nature of pus ; it becomes whiter and whiter, losing more and more of the yellow and green, which it is apt to give the linen that is stained with it in its first stages, and in consistence more and more viscid or creamy."

" Pus is not to be found in the blood, similar to that which was produced in the first stage ; but is formed from some change, decomposition, or separation of the blood, which it undergoes in its passage out of the vessels, and for effecting which, the vessels of the part have been formed."

He further adds, " we must look upon it as a new combination of the blood itself, and must be convinced that in order to carry on the decompositions and combinations necessary for producing this effect, either a new or peculiar structure of vessels must be formed, or a new disposition, and of course a new mode of action of the old must take place. This new structure, or disposition of vessels, I shall call glandular, and and the effect or pus, a secretion." It may be remarked here, that this glandular structure, to which the French pathologists give the name of *tissu pyogénique*, is by no means necessary to the formation of pus.

From this general statement of the views entertained by Mr. Hunter on the formation and nature of pus, it will appear obvious that he considered suppuration, wherever it occurred, as always a consequence of inflammation ; that the vascular structure of the part underwent a certain change, in consequence of which there were separated from the blood, by a process similar to that of secretion, the materials which constitute pus ; that this fluid is gradually formed during the successive changes which characterize the inflammatory state ; that is to say, there succeeds to vascular congestion, first, the effusion of serum, afterwards coagulable lymph, and lastly pus, all of which are often discharged in succession, more especially from the surface of wounds and of serous cavities. Although it does not appear that Mr. Hunter ascertained the particular changes which take place in the blood of the capillaries of an inflamed part, during the suppurative stage, it is clear that he considered pus as essentially composed of the constituent elements of the former fluid ; for, when speaking of the discharge which accompanies the suppurative stage, he thus emphatically expresses himself : "*this matter is a remove further from the nature of the blood, and becomes more and more of the nature of pus*"—that is to say, it is only a remove further from the nature of the blood than the matter formed by adhesive inflammation,—an opinion, the accuracy of which has received the fullest possible confirmation from the recent experiments of Gendrin, who expresses himself on this point in nearly the same language : " Il n'y a entre le fluide purulent des tissus enflammés et le fluide coagulable organisable qu'un degré de plus." I shall only further remark in reference to the services rendered by Mr. Hunter to the science of pathology by his researches on this particular subject, that he contributed more than any other author to the overthrow of the old doctrines regarding

suppuration, the nature and effects of pus; and that, having established the intimate relation which exists between the process by which pus is formed, and secretion,—a vital or physiological act of the economy,—he pointed out to succeeding enquirers the only mode of investigating this subject successfully.

Before proceeding to describe the properties of pus, I shall endeavour to give a clear and concise view of the process of suppuration, when a consequence of inflammation, occurring in parts in which the phenomena which it presents can be distinctly seen, and submitted to the means we possess of determining their nature.

Formation of Pus.—The capillary vessels of the transparent parts of animals, which can be conveniently placed under the field of the microscope, are best calculated for exhibiting the changes which take place in the blood, or the fluids which are separated from it during the formation of pus.—When inflammation has been excited in the web of the frog's foot, that is to say, when the capillary vessels have become distended with red globules which have ceased to circulate, the following phenomena are observed to follow:—the capillaries present an almost uniform red colour; serosity, coagulable lymph, and, if the inflammation has been severe, blood, are effused in succession into the surrounding cellular tissue; the whole of the inflamed part becomes somewhat opaque; by-and-bye the red colour of the blood begins to disappear in various points, and a yellow-coloured granular-looking matter occupies the capillaries and the interstitial cellular tissue; in the central portion of the inflamed tissue several of the capillary vessels which were before obscured by the accumulated blood, begin to re-appear, some containing reddish, others yellowish grey globules, which gradually become more distinct, increase in number and size, begin to move slowly, and traversing the capillaries, arrive at the surface of the tissue, or at the edges of the solution of continuity if this has occurred, in the form of globules of pus.

Gendrin, who has carefully investigated, under a great variety of circumstances, the changes which take place in inflamed tissues, gives a minute, and, I believe, an accurate description of the process of suppuration. He excited inflammation in the web of the frog's foot and in the mesentery by means of boiling water, the actual cautery and the seton, and followed with the microscope the gradual development of pus from the globules of the blood. He relates as having distinctly seen the globules of the blood, after having been brought to a state of stagnation in the capillaries, becoming deprived of their colouring envelope and opaque, and assuming a yellowish grey colour approaching to that of pus; that he has then traced them moving slowly in the capillaries or in new-formed vessels, and as they advanced towards the edge of the ulcer or eschar occasioned by the violence of the inflammation, gradually acquiring all the physical characters of perfect pus. Kaltenbrunner, than whom there does not appear to have been a more able, cautious, and philosophical observer, had, before the researches of Gendrin were published, arrived at nearly the same results. The microscopical researches of this author, however, would seem to shew that not only is the blood which is carried into the inflamed tissue, but likewise a portion of the solids, converted into pus; for he states that small granular bodies are seen to separate from the *parenchyma*, to pass into canals which are formed for their reception, and to mingle with others of a similar kind coming from the blood, both of which are gradually converted into true granules of pus.

From the very obvious character of the facts elicited by these microscopical observations, no doubt can be left in the mind that the formation of pus is a consequence of a modification of the blood, manifested more especially by a change

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taking place in the colour, transparency, and bulk of the globules of this fluid, after its circulation has been arrested in the capillaries by inflammation; that this change in the globules takes place in the capillary vessels, and that these vessels conduct the globules in this state to the exterior, where they appear combined with the serum of the blood under a peculiar liquid form, or that which we call pus.

Such is one mode in which pus is formed, that, in fact, which has been likened to the process of secretion. There is, however, another mode, which, to a greater or less extent, is, perhaps, always in operation at the same time with the former, but which, under certain circumstances, is effected alone, and from its not having been properly understood, has created much confusion, and given rise to opinions the most repugnant to the principles of a sound and philosophical pathology: I allude to the formation of pus in the blood, under circumstances in which the influence of the capillary system, as exercising a function of secretion, can have no part. Some evidence has already been adduced in favour of this mode of formation of pus, and which may be called *extra-vascular*. Thus we have seen, that besides the conversion of the globules of the blood, contained in the capillaries, into pus, there was also observed a similar change in those of the blood effused into the cellular tissue around these vessels; in fact, the whole of the blood, *intra* and *extra-vascular*, was seen to undergo the same gradual change of colour from red to yellowish grey; the globules in both situations became opaque, acquired an increase of bulk, and passed slowly towards the surface of the part, either in the original or new-formed capillary vessels in the form of pus. In every case of violent inflammation of the cellular tissue for example, the increase of bulk and the induration by which it is followed, are owing, in a great measure, to the presence of effused blood. A section of the part shews this distinctly, whilst at the same time the capillaries, minute arteries, and veins are distended with the same fluid. When suppuration supervenes, the gradual discolouration, softening, and conversion of the whole into pus, succeed to each other, and must be produced on the same principles as those observed in the blood of a single capillary under the microscope, for the pus is equally perfect in both cases. I have, on several occasions, been able to satisfy myself that this mode of formation of pus often occurs to a considerable extent in blood effused into the cellular tissue, from external violence, and when followed by acute inflammation; but I have most frequently observed it in the blood which has ceased to circulate in inflamed veins. Thus, in the first case, the conversion of the blood into pus is proved by this latter fluid alone being found some time after in the swelling which had been originally produced by the effused blood. Gendrin has put this fact beyond all doubt by direct experiment. "If, after having injected," observes this author, "a great quantity of blood into the subcutaneous cellular tissue, a seton is passed through the same tissue in order to excite a certain degree of inflammation, the blood is rapidly converted into pus, as if it had escaped from the vessels themselves of the part."

In phlebitis again, the conversion of the blood into pus can often be most satisfactorily demonstrated. As soon as inflammation of a vein has acquired a certain degree of severity, one of the most remarkable circumstances which attracts our notice, is the cord-like hardness of the vessel. The blood has ceased to circulate through it—it has coagulated. At a subsequent period, however, we find that this state of the vessel becomes less conspicuous, and disappears, sometimes suddenly, no cord-like swelling or induration being afterwards felt. Now, further examination shews that the latter occurrence was the result of the blood having been converted into pus. The

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almost immediate consequence of phlebitis of a certain extent being the coagulation of the blood in the affected vessel, it therefore follows that if the same vessel is afterwards found filled with pus, the blood thus coagulated, and which could not have been carried into the circulation as such, must have been transformed into pus. The accuracy of this conclusion is further illustrated by the appearances found on dissection of a limb affected with phlebitis in the inflammatory and suppurative stages. The blood can be traced, sometimes in the same, sometimes in different veins, to have undergone a gradual change into pus. In the veins which are but slightly inflamed, it is found in a state of fluidity; in those in which the inflammation is marked by great vascularity, serous and albuminous effusion in the middle and external coats, it is coagulated, slightly adherent to the internal membrane, or united to it by a thin layer of unorganized coagulable lymph; in the former it presents the dark red colour of venous, in the latter, the bright red colour of arterial blood. As we approach those portions of the veins in which the inflammation commenced, or in which it has proceeded to the suppurative stage, the arterial colour of the blood passes gradually into a pale yellow or that of fibrine, of which it has at first also the consistence, but becoming soft, pulpy, and creamy, until it is at last lost in the puriform matter contained in the vessels or in the surrounding tissues. These views respecting the conversion of the blood into pus in inflamed vessels of large dimensions, are fully confirmed by the following ingenious and interesting experiment of Gendrin. "If, having interrupted for an instant," says this author, "the circulation in an artery or vein, a solution of nitrate of silver or caustic potash be injected, and soon after withdrawn, and the blood again admitted and retained in the vessel by means of two ligatures, suppuration ensues; the blood, at first coagulated, afterwards becomes discoloured, and is progressively converted into pus." "This conversion of the blood into pus," he adds, "can be followed by the eye between the layers of the coagulum, and by the microscope in the molecular or globular structure of the fibrine."

From all these facts it must be evident that the capillaries could have had no share in the purulent transformation of the blood in the examples which have been brought forward of this change, and therefore it follows that the formation of pus cannot be restricted to a morbid process induced in these vessels by inflammation, and the subsequent separation of the elements of this fluid from the blood. In these experiments and observations there are, however, three striking circumstances which, from their constancy, the regularity of their occurrence, and the uniformity of their results, must be regarded as having a common origin, or as consequences the one of the other: these are, the cessation of the circulation, the coagulation of the blood, and the conversion of the fibrine or globular structure of this fluid into pus. These three series of changes we have always seen succeeding to each other, that is to say, the globules of pus made their appearance in the fibrine of the blood which had become deprived of its colouring matter after having been brought to a state of stagnation in the capillaries, in arterial and venous trunks, and in lacerated excavations formed in the cellular tissue. But we have also seen that these changes were always preceded by inflammation of the tissue in which the blood was contained, and the pus that was immediately derived from this fluid. That inflammation was the common origin of these changes cannot, therefore, admit of a doubt; and as it has been proved that they constitute the process of suppuration, the legitimate conclusion at which we arrive is, that this morbid process is essentially dependent upon inflammation as its efficient cause.

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Although this view of the nature of suppuration be in accordance with the opinions of the greater number of modern pathologists who have investigated this subject, it has of late years been considered by many as altogether insufficient to explain the formation of certain kinds of abscess, but more especially the presence of purulent deposits or infiltrations, as they are called, which occur in various parts of the body without being accompanied by the usual characters of inflammation. Hence has arisen the question, is the presence of pus always to be considered as a product of inflammation of the part in which it is found? Much of the difficulty which has been encountered by those who have sought a solution of this question, and much of the confusion which has prevailed in their writings, would, I conceive, have been avoided, had the pathological principles which I have endeavoured to establish been previously understood, and a distinction drawn between the process of suppuration, considered as a vital act, and the mere presence of pus as a product of that process. If pus is found in an organ in which neither the physical nor physiological characters of inflammation are to be detected, either during life or after death, the necessity of establishing a distinction between the *mere presence of pus* and *suppuration* must be obvious. That pus is formed under such circumstances is what I shall now endeavour to prove; first, from this fluid being found in the blood where inflammation could have exercised no direct influence in its production; secondly, from its being found in organs under the circumstances stated above. The most conclusive evidence that pus may present itself simply as a foreign body in the blood, is obtained from the fact that it has been frequently found in coagula contained in the cavities of the heart. I allude particularly to cases of this nature which I have met with, in which there was no trace of disease of any kind of this organ. The coagula were not adherent to the internal membrane of the heart; they were retained by prolongations interwoven with the columnæ carneæ; their consistence varied from that of a recent clot to that of firm fibrine, and their colour was partly of a deep, partly of a bright red tinged with yellow. The pus which they contained appeared in minute points, striæ, or drops, varying from the size of a pin's head to that of a grain of hemp-seed; it was of the consistence and colour of healthy matured pus, and occupied the central portions as well as the surface of the coagula, but having a tendency to accumulate in the form of striæ. This latter circumstance appeared to originate in the laminated and fibriliform arrangement which the fibrinous portion of the coagula had assumed, the globules of pus being thus disposed of in rows or punctiform lines, apparently after its separation from the blood, by the spontaneous coagulation or contraction of the fibrine. That cases of this kind are examples of the formation of pus without the existence of inflammation of the heart, and consequently beyond the influence which this pathological state is known to exercise in the production of this fluid, cannot for a moment be called in question. Andral, Reynaud, and other French pathologists have recorded examples of a similar kind, and we shall find that, were we not to admit the fact that pus is actually formed under such circumstances, it would be impossible to offer anything like a rational explanation of the occurrence of those puriform deposits to which I have alluded. But there is another most important fact which requires to be established before we can understand how pus can be formed in coagula in the cavities of the heart, and that is, the co-existence of suppuration in some other organ. I have never met with a case of anomalous formation of pus, either in the cavities of the heart, in the cellular or parenchymatous structure of organs, or in the cavities of serous membranes, without finding at the same time inflammation

and suppuration to a greater or less extent in some remote organ. It may indeed be asserted that this is also the almost uniform result of the researches of other pathologists, although Andral, Maréchal, and some others have rather vaguely stated that they have met with puriform matter in fibrinous concretions of the heart, without the presence of pus being detected in any other organ of the body. Such cases, several of which I have also seen, are very different from those now under consideration. The fluid matter of these concretions, so far as my observation goes, never resembles pure pus; it is a thin, grumous, grey or reddish coloured fluid, but probably puriform in its nature, as it resembles the contents of those concretions which are formed during life in the cavities of the heart, in some cases of inflammation of the internal membrane of this organ, succeeding to rheumatism of the joints. If such be the origin of puriform collections of this kind, they of course come under the head of suppuration, and do not form an exception to the law, that the formation of pus in the blood and in the other parts of the body which I have enumerated, under circumstances which disprove its connexion with inflammation as cause and effect, has never been shewn to take place without being preceded by suppuration in a remote organ. This important fact being admitted, let us pass in review the principal circumstances under which suppuration has been observed to precede the occurrence of these anomalous formations of pus. These circumstances are, external suppurating sores; wounds; operations, such as those of amputation, of lithotomy, of fistula in ano; the resection of portions of diseased bone; fractures; phlebitis occurring idiopathically, after bloodletting, wounds, or other external injury, more especially of the extremities; and after delivery, in the uterus and its appendages. In all these cases suppurative inflammation had taken place to a greater or less extent,—a circumstance which, considered in relation to the etiology of purulent deposits, will be found to possess all the importance which has been attached to it.

The situation of the pus in these cases of suppurative inflammation is another circumstance which deserves particular notice. In the great majority of cases this morbid product is found in the veins of the affected part. In idiopathic phlebitis, in phlebitis of the uterus following delivery, and even in phlebitis after bloodletting, it is sometimes found only in the veins; whereas, in phlebitis succeeding to external injuries or operations, it exists, often extensively, at the same time, in the intermuscular cellular tissue, in the canals and cancelli of the bones. It is found in the smallest veins that can be traced by dissection, in the largest trunks of the extremities, and pelvic viscera, and even in the vena cava. In some cases the quantity of pus does not amount to a drachm, in others it measures several ounces. The number of veins in which it is found varies from a very few, small or large, to the whole of those of the arm or forearm, or of the uterus. The arteries, in such cases, never contain any pus, nor have I met with it to any extent in the lymphatics, except in uterine phlebitis, in some cases of which these vessels were distended with it, the veins being in a similar state, or containing only a very small quantity.

The description which I have already given of the mode of formation of pus in inflamed veins, renders it unnecessary to describe the appearances which the blood and pus present in these vessels. As the blood which becomes coagulated in such cases is converted into pus, it is impossible to say how much of the latter has been derived from this source or from secretion. That it is not derived from absorption will appear more than probable presently. A circumstance, perhaps, more deserving of attention is the relation which is found to exist between the pus in the inflamed and the blood in the neighbouring

healthy veins. In this respect, the pus appears, 1st, to have been either entirely prevented by a cylinder of firm fibrine, adhering to the internal membrane of the inflamed vein, or partially, by a loose but defined coagulum of dark blood, from passing into the neighbouring healthy veins; 2dly, no obstacle exists which could have prevented a communication from taking place between the pus in the inflamed, and the blood in the healthy veins. I have certainly not seen a case in which this communication was not more or less extensively apparent. With regard to the veins themselves which contain the pus, it is only necessary to state that they present all the physical characters of acute inflammation to a greater or less extent, such as various degrees of increased vascularity of the middle and cellular coats, accompanied with the effusion of coagulable lymph, softening, sometimes perforation and a state of sphacelus.

With all these facts before us, viz.—the existence of suppurative inflammation; the presence of a greater or less quantity of pus in the veins; evidence that the pus so situated is the product of inflammation of these veins; and that this morbid product is carried into the blood by the collateral venous circulation;—it appears to me that a satisfactory explanation may be given of the formation of those anomalous collections of pus, to which I have hitherto alluded, which take place in remote parts of the body. Let it be remarked, however, in the first place, that, whilst all the pathologists who have studied this subject, concur in the opinion that the pus is taken into the circulation, they differ materially as to the manner in which they suppose this to be accomplished. By some—Velpéau, Maréchal, Rochoux,—it is attributed to absorption from suppurating surfaces; by others,—Dance, Blandin, Cruveilhier,—simply to the transmission of the pus from the veins in which it is formed. They agree also in the opinion that the pus taken into the circulation is the cause of the purulent deposits. These, according to the former authors, are formed as a consequence of the separation of the pus from the blood, and its subsequent accumulation in the capillaries or the cellular texture; according to the latter, they are the result of suppurative inflammation induced by the pus acting as a foreign body, either in the capillaries, in which case suppuration takes place as under ordinary circumstances; or in the smaller veins, when it is produced in consequence of phlebitis.

With regard to the first of these opinions, viz. that the pus is carried into the circulation by absorption, it is only necessary to observe that, besides the incomplete and unsatisfactory nature of the evidence in support of this opinion, it is one which we are at once led to reject on the very obvious ground, that absorption is not required to explain the introduction into the circulation of pus which we find to have been formed, if not in all, at least in the great majority of cases, *ab origine* within the veins. Some of the worst forms of purulent deposition occur in those severe cases of phlebitis occasioned by mechanical injury or otherwise, in which the pus is formed in the veins, and from them passes directly into the circulation. It is, therefore, not necessary to have recourse to absorption to explain the introduction of pus into the circulation; and as an argument against this being the source of purulent depositions, it may be remarked, that abscesses and other collections of pus often disappear by means of absorption, without being followed by any of the local or general effects which are so conspicuous and so fatal in suppurative inflammation of the veins.

With regard to the two opinions which I have stated are now entertained as to the manner in which the purulent deposits are formed, I think there is sufficient evidence to prove that they are both supported by numerous facts. We have already seen that such deposits take place in coagula formed in the cavities of the heart, and it would be diffi-

cult to assign a reason why they should not take place in other organs, if circumstances occurred to interrupt the circulation through the capillaries. It is possible that the pus itself might effect this change in the capillary circulation, and occasion those circumscribed congestions so conspicuously seen in the lungs, and which constitute the first stage of the purulent deposits in these organs. The formation of the pus in these cases can be seen to take place in the same manner as in the coagula in the cavities of the heart. The blood with which the capillaries are distended, and which is sometimes effused at the same time into the surrounding cellular tissue, is coagulated, of a deep red colour, and forms globular masses varying from the size of a pea to that of a walnut, of considerable density. The first appearance of the pus in these masses is recognised by the presence of a number of pale yellowish grey points, which increase in number and bulk until the whole of the blood is converted into a substance resembling in colour and consistence the fibrine of the blood. After this, the second stage is observed to follow the conversion of the fibrine into pus, and the formation of an abscess, the size of which is determined by the extent of the previous congestion. In some cases, however, the formation of the pus does not appear to be preceded, in the manner just described, by the separation of the fibrine into a solid mass. It is occasionally found in drops in the coagulated blood, and therefore bearing a stronger analogy to what we have seen takes place in coagula in the cavities of the heart. A similar mode of formation of the purulent deposits has been observed in the subcutaneous and intermuscular cellular tissue, in the spleen and in the liver. In this latter organ, however, these deposits present an appearance peculiarly characteristic of their origin, and which will be described in the plates in which they are represented. To these facts in support of the opinion that purulent deposits are formed by the separation of pus from the blood, may be added those which disprove the existence of inflammation as their efficient cause; such as the absence of vascular congestion, of coagulable lymph, induration or softening of the surrounding tissues, and of any appreciable modification of function of the affected organ, more especially of such a nature as accompanies this pathological state, when it terminates in suppuration to the extent which the purulent deposits frequently occupy.

The opinion that the purulent deposits are the result of suppurative inflammation, induced in remote organs by the pus circulating in the blood, appears to me to rest also on the most conclusive evidence. In many cases, we meet with all the physical characters of inflammation; and, besides, the tissues in contact with the pus are softened, ulcerated, or covered with layers of coagulable lymph. There are sometimes pain, heat, and swelling in the joints in which the pus is found after death, and also in the situation of abscesses formed in the intermuscular and subcutaneous cellular tissue; and well-marked symptoms of pleurisy precede the formation of similar collections of pus in the cavity of the chest. Indeed we very often find coagulable lymph on the pleura, where it covers the purulent deposit in the lungs. I have stated that the inflammation which gives rise to the present form of purulent deposition is said by some pathologists to have its seat in the minute veins; that in fact these deposits not only have their remote origin in phlebitis, but that they are likewise the immediate consequence of the same morbid state. I have certainly not been able to satisfy myself of the accuracy of this opinion in cases of purulent deposits in the lungs or liver; and that it applies to those which form in the serous and synovial membranes, cannot be admitted, unless phlebitis and inflammation are considered as synonymous terms.

In terminating this part of the present subject, it must not be overlooked that the

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theory which attributes the formation of purulent deposits to the presence of pus circulating with the blood, derives strong corroborative evidence from the peculiar character of the general symptoms which occur towards the termination of this fatal disorder. The sudden, and, frequently, unexpected occurrence of great disturbance of all the functions of the economy, often at a time when the patient appears to be out of all danger, characterized principally by great prostration of strength, confusion and stupor of the intellectual faculties, a dingy yellow colour of the skin, fætor of the breath, meteorism and petechiæ, constitute the more remarkable changes which accompany the formation of purulent deposits; and when we compare these with the analogous effects, both local and general, produced by the injection of pus and putrid fluids of various kinds into the veins of inferior animals, we are compelled to admit that the evidence which we possess in support of this theory is equally satisfactory and complete.

In order to complete the history of purulent deposits, it may be remarked that the number of organs in which they occur, varies considerably. In some cases only one organ is affected, sometimes to a very small, sometimes to a great extent; whilst in other cases a greater or less number of organs are affected in a similar manner. The lungs and liver are far more frequently affected with these deposits than any of the other organs of the body, whatever may be the seat of the lesion in which they originate. Recent observations shew that those of the liver are not more frequent after injuries of the head than of the extremities. Of all the organs of the body the kidneys are least frequently the seat of purulent deposits. The few cases which are recorded as examples of this kind, appear to me to have been the result of inflammation extending to the kidneys from neighbouring organs, and succeeding to the operation of lithotomy, to injuries of the spine, to the presence of calculi, and to various diseases of the pelvic viscera. We cannot, perhaps, appreciate the importance of this circumstance, but it is extremely probable that it is to be accounted for by the separation of the *material* cause of these deposits from the blood carried into these organs, and its excretion along with the urine.

Having devoted the greater part of the present fasciculus to the illustration of what appeared to me to constitute the most important part of the pathology of suppuration, I shall confine myself, in the further consideration of the subject, to a few general observations on the properties of pus.

Properties of Pus.—There is, perhaps, no morbid product which has been so often submitted to the analytical researches of the pathologist, as pus; notwithstanding, the results of these researches possess little or no value as regards the object for which they were chiefly prosecuted, that of ascertaining the presence of certain properties in this fluid by means of which it might be distinguished from mucus in diseased states of the tissue which furnishes this secretion. It is, indeed, surprising that any, or at least, much importance should have been attached to this circumstance, after it was known that the formation of pus has no necessary connexion with ulceration, or that mucous and serous tissues, which have undergone no solution of continuity, furnish this fluid in great abundance. All the observations which have been made on pus with this view may therefore be passed over in silence, and the pathologist safely left to estimate the nature of the changes which take place in the secretion of mucous canals, by his knowledge of the physical characters of pus, and the pathological condition on which its presence essentially depends.

Physical properties.—Pus, *pure* or *laudable* pus, as it is called, such as we find it on the surface of a healthy granulating sore, consists of a homogeneous fluid, of the con-

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sistence of cream, heavier than water, of a yellowish white colour, somewhat tenacious, having an insipid taste, and a mawkish smell when warm. When examined in the microscope, it is seen to be composed of a multitude of globules floating in a transparent fluid, the relative proportions of which vary considerably. The globules are opaque, of a slight yellowish tint, and larger than those of the blood; and it would appear that it is to their presence in the fluid, that the colour and consistence of pus is to be attributed, the degree of which, particularly the latter, varying with their number. Although the fluid resembles the serum of the blood, it is found to differ from it in being coagulable by the muriate of ammonia.

Chemical properties.—Formed by a process similar to that of secretion, the chemical composition of pus must vary, not only with the nature of the tissue from which it is immediately derived, but likewise under the influence of various morbid conditions which are known to modify the products of secretion in general. It is on this principle that we explain the difference which exists, particularly at the commencement and termination of suppuration, between the pus furnished by serous and mucous membranes, the quantity of albuminous matter being much greater in the former than in the latter case; and, as has been observed by Gendrin, that the pus furnished by the granulations in caries contains a greater quantity of the phosphate and muriate of lime; the puriform discharge of scrofulous ulcers a larger proportion of soda and the muriate of soda; and that which is found in the tissues surrounding the joints in gout, an excess of the carbonates, phosphates, and, perhaps, the urate of lime. A difference in the degree of the inflammatory excitement of the affected part; the supervention of an acute disease in a remote organ; a sudden emotion, whether followed by great excitement or depression, &c., produce also obvious changes in the physical, if not in the chemical characters of pus. The several varieties of pus which are formed under these or other modifying causes, have received from Mr. Pearson the following appellations;—1. creamy pus; 2. curdy pus; 3. serous pus; 4. slimy pus; to which Andral has added a fifth, concrete pus, which is the same as the curdy of Mr. Pearson, but deprived of a great part of its serum by absorption, such as it is often met with in scrofulous abscesses. They are found to be composed of albumen in a state of concretion, water, extractive matter, a substance resembling adipocire, and various salts. Chemical analysis, therefore, does not establish any important distinction between pus and the serum of the blood, the difference, in this respect, between the two fluids consisting in the state of concretion of the albumen, and a modification of the extractive matter.

Specific properties.—With regard to the specific properties of pus, I have only to remark, that they have received no elucidation from any mode of investigation to which they have been subjected. The pus of small-pox or of a syphilitic ulcer differs in no respect in its physical and chemical properties, from that of a phlegmon. The specific properties, of which the pus in the former is merely the vehicle, are known only by their operation on the living body.

DESCRIPTION OF THE PLATES.

PLATE I.

Fig. 1 represents a rare form of abscess of the brain ; it resembled the chronic abscess of cellular tissue, in the pus being contained in a membrane analogous in its structure to mucous tissue. There were two abscesses of this kind in the left hemisphere ; one of them, A, sufficiently large to contain a small hen's egg, the other, B, capable of receiving only the point of the little finger. They were both filled with thick, greenish pus ; a portion of which in the large one was concreted into a thin layer A, of the consistence of firm cheese, which adhered slightly to the mucous tissue, a portion of which, B, has been exposed by the removal of a small quantity of the former. *Fig. 2* represents purulent deposition and abscess of the brain, succeeding to extensive suppuration of the hand and forearm ; A, the cerebral substance broken down, mixed with pus, and presenting here and there streaks of blood ; B, a large portion of the same substance infiltrated with this fluid and nearly detached. *Fig. 3* is a remarkable example of suppuration of the grey substance of the two lateral halves of the medulla spinalis. It originated in inflammation of a limited portion of the membranes occasioned by caries of the spine. A, pons varolii ; B, medulla oblongata ; C, C, comprehends a longitudinal section of the cord, exposing the whole extent of the suppuration of the grey substance ; D, the primary seat of the inflammation of the cord, which afterwards extended downwards on the left side in the direction of the grey substance towards E, and upwards towards F, where it crossed to the right side and proceeded again upwards as far as G, still keeping in the situation of the grey substance H ; from E, on the left side, it stretches as far up as K, the commencement of the medulla oblongata. An albuminous, granular exudation, M, covers the membranes of the cord in the original situation of the disease. *Fig. 3, a, b*, represents sections of the cord to shew more distinctly the pus contained in the grey substance ; in the section a, the suppuration is seen proceeding from the inflamed portion of the external surface of the cord to the grey substance. *Fig. 4, A, A*, the kidney containing a great number of small abscesses, the result of inflammation of the cortical substance, succeeding to suppurative inflammation of the prostate, and stricture of the urethra ; B, great dilatation of the ureter from retention of the urine. *Fig. 5*, purulent deposits in the intermuscular cellular tissue of the heart ; A, a section of the parietes of the left ventricle ; B, a collection of several small deposits.

Fig. 6, small-pox pustules in the larynx, trachea, and bronchi. The pus was situated in the mucous tissue; in some parts, as on the inferior surface of the epiglottis A, it presented a diffuse form; in others, as in the larynx B, in portions of the trachea C and D, and bronchi E, it assumed the circumscribed pustular form, giving rise to that arrangement of pustule observed in discrete and confluent small-pox. The mucous membrane of the larynx, but especially of the trachea and bronchi, was of a uniform deep red colour. The patient, a female, was 30 years of age.

PLATE II.

Fig. 1, purulent deposits in the lungs, succeeding to suppurative inflammation of the medullary membrane and veins of the femur, after amputation. A, lower, B, upper, C, middle lobe of the right lung; D, D, circumscribed, deep red, indurated portions of pulmonary tissue, representing the first or congestive state of purulent deposits in the lungs; E, E, and F, F, the appearance of granular points gradually assuming the colour and consistence of pus, during the second stage; G, G, represents the completion of this process, or the entire conversion of the coagulated blood, into a yellow puriform substance, having the form of round masses of considerable size; H, H, these masses assuming the form of abscess. The whole of the inferior lobe was highly congested, but there was no pus formed except in those portions of it in which the coagulation of the blood had taken place. *Fig. 2* represents a small portion of lung, A, from a similar case. It presents no appearance of congestion, and no trace of disease except the presence of pus in a vein B, of considerable size, and which occupies its minutest divisions. *Fig. 3* represents purulent deposits in the lungs succeeding to extensive suppurative inflammation and sloughing of the integuments and muscles situated over the sacrum. In one portion of the lung only, A, is seen an indurated, granular mass of coagulated blood, which is gradually converted into smaller masses of a pale yellow coloured fibrinous matter B, and this again into creamy pus C, contained in a vein of the second or third order, and in contact with a small pale coagulum D, the vein, at this part, and towards its termination E, presenting its natural colour and consistence. The complete conversion of the blood into pus and the formation of abscess are represented at F and G. *Fig. 4* represents suppurative inflammation of the pulmonary tissue terminating in abscess, and the cellular sheath of the veins infiltrated with pus:—this circumstance might be overlooked, and the veins described as containing pus. The cellular sheath of the veins is generally in this state in erysipelas phlegmonodes, and also in the first stage of phlebitis. A, walls of the abscess; B, cavity of the vein exposed; C and D, the cellular sheath of a straw yellow colour and infiltrated with pus. *Fig. 5* represents purulent deposits in the liver, succeeding to extensive suppuration of the arm. Section of a large abscess A, situated in the right lobe, the walls of which, formed of condensed cellular tissue, are infiltrated with pus; C, C, several puriform deposits not yet softened, and surrounded by a greenish grey areola. *Fig. 6* represents the same lesion in its several stages, as it usually occurs in this organ, and succeeding to a blow on the head. The purulent deposit is seen at A A, to be composed of a group of oval or circular bodies, each having a central depression or slit; these are the acini of the liver, enlarged and transformed into a straw yellow colour by the accumulation of pus, probably contained in their vascular structure. A similar appearance is

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seen on the surface of the liver at B. The purulent deposit in this the first stage is of the consistence of firm fibrine, and terminates by an abrupt margin, having an irregular outline, in consequence of its being formed by the enlarged acini. At the commencement of the second stage, the deposit loses the appearance which it derives from the enlargement of the acini; it assumes a more uniform aspect, becomes soft, and is transformed into a number of irregular excavations, C, C, C, filled with pus, sometimes mixed with sloughs or a small quantity of blood; the complete softening of the deposit constitutes the third stage or that of abscess, so conspicuous in *Fig. 5*. Purulent deposits in the liver are frequently surrounded by a dark, dirty green areola, as shewn in the present *fig.*, and which is accompanied with a gangrenous odour, or that of putrefaction.

PLATE III.

Fig. 1, 2, 3, 4, and 5, represent suppurative inflammation of the medullary canal of the femur and phlebitis. *Fig. 1*, a longitudinal section of the upper portion of the femur, after amputation of the thigh; the inferior half of the medullary canal is filled with fibrinous clots and pus, A, A; the cancellated structure of the bone B, B, contains also a considerable quantity of pus, and in some parts, C, is much injected; the extremity of the bone, D, presents the grey discolouration of necrosis; the periosteum E, which was softened, is detached, and on some parts, F, covered with a puriform matter. *Fig. 2* represents the femoral vein laid open, which throughout its whole extent, A, B, was filled with pus, and lined with a pale, yellow grey, soft, pseudo-membranous substance, extending to the surface of the stump C, which was of a dirty blueish grey colour and emitted a gangrenous odour; D, D, two branches of the femoral filled with pus; two other branches filled with coagulated blood; the coats of the femoral vein were greatly thickened and indurated, and those of the branches which contained pus were much injected. *Fig. 3* represents a more advanced stage of the same disease of the medullary membrane of the femur following amputation; the medullary canal is transformed into a large abscess A, whilst the cancelli, B, B, B, of the upper portion of the bone, are distended with fibrinous clots, concrete and fluid puriform matter; the walls of the bone, C, C, C, are thickened and partially necrosed; and the periosteum D separated. *Fig. 4* and *5* represent what is frequently observed in the first stage of phlebitis: the cellular sheath A, *fig. 4*, is highly injected, thickened, and of a red colour throughout its whole depth B; whilst the middle coat is infiltrated with pus, which gives a yellow colour to the internal membrane C; which is sometimes thrown into longitudinal folds, as represented at B, *fig. 5*, and terminating abruptly at C, by a serrated border. *Fig. 4, a, b*, represents the same state of the vein as seen in a transverse section. It sometimes happens, when the inflammation extends from the surrounding cellular tissue to the vein, that the vein is compressed, and the circulation impeded or interrupted before the effusion of pus into its cavity takes place, as happened in this instance; the small coagulum represented in *fig. 4*, is situated in the healthy portion of the vessel. *Fig. 6*, a section, A, of the spleen, containing a small mass of concrete pus, B, in communication with a vein. *Fig. 7*, coagula contained in the left ventricle, A, A, of the heart; B, origin of the aorta; C, C, C, points, striæ and drops of pus in several portions of the coagula; D, a portion of the mitral valve.

PLATE IV.

Fig. 1. Uterine phlebitis ; A, A, large drops of pus projecting from the orifices of the veins on the cut surface of the uterus ; several of the orifices seen at E ; B, the neck ; C, the fundus of the uterus ; D, the vagina ; the two former of a dark blueish grey colour ; they were softened, and emitted a gangrenous odour ; the Fallopian tubes F, F, highly inflamed, and containing a puriform matter ; G, G, the ovaries, and H, H, the peritoneal surface of the uterus covered with a layer of coagulable lymph. *Fig. 2,* a diminished view of the uterus of a young woman who died the seventh day after delivery, having presented the usual symptoms of uterine phlebitis. There was, however, no trace of this disease, except at the upper and posterior part of the vagina, where only one small vein contained a few drops of pus ; but the lymphatics of the uterus, broad ligaments, and Fallopian tubes were distended with pus. These vessels, I believe, absorbed it from the surface of the uterus, which was covered throughout its whole extent with a yellowish grey pultaceous matter, resembling a mixture of pus and fibrine, beneath which the substance of the uterus was red, vascular, and very soft : A, the cut surface ; B, the fundus ; C, the neck of the uterus ; D, vagina ; E, E, E, the Fallopian tubes, swollen and injected ; F, F, their fimbriated extremities in a similar state and covered with coagulable lymph ; G, the right ovary greatly enlarged, and also covered with lymph ; H, the left ovary injected with lymph, and containing cells, filled with coagulated blood : K, K, the lymphatics of the broad ligaments and Fallopian tubes greatly enlarged and filled with pus, and proceeding upwards along the spermatic veins M, M, and terminating in two lymphatic glands Q and R. N, the inferior cava ; P, the right emulgent vein.

Plate I.

Fig. 1.



Fig. 3.

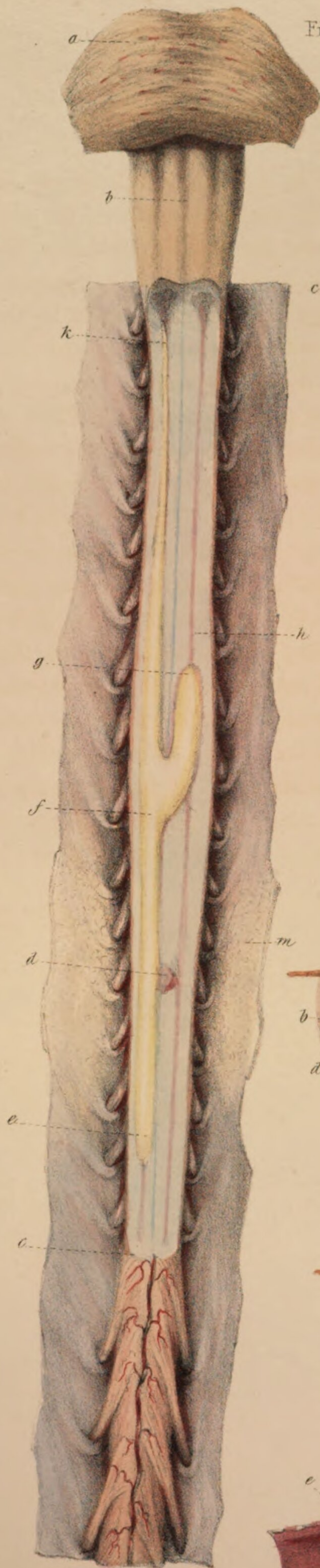


Fig. 2.



Fig. 5.



Fig. 6.



Fig. 4.



Fig. 3.



Plate II.

Fig. 1.

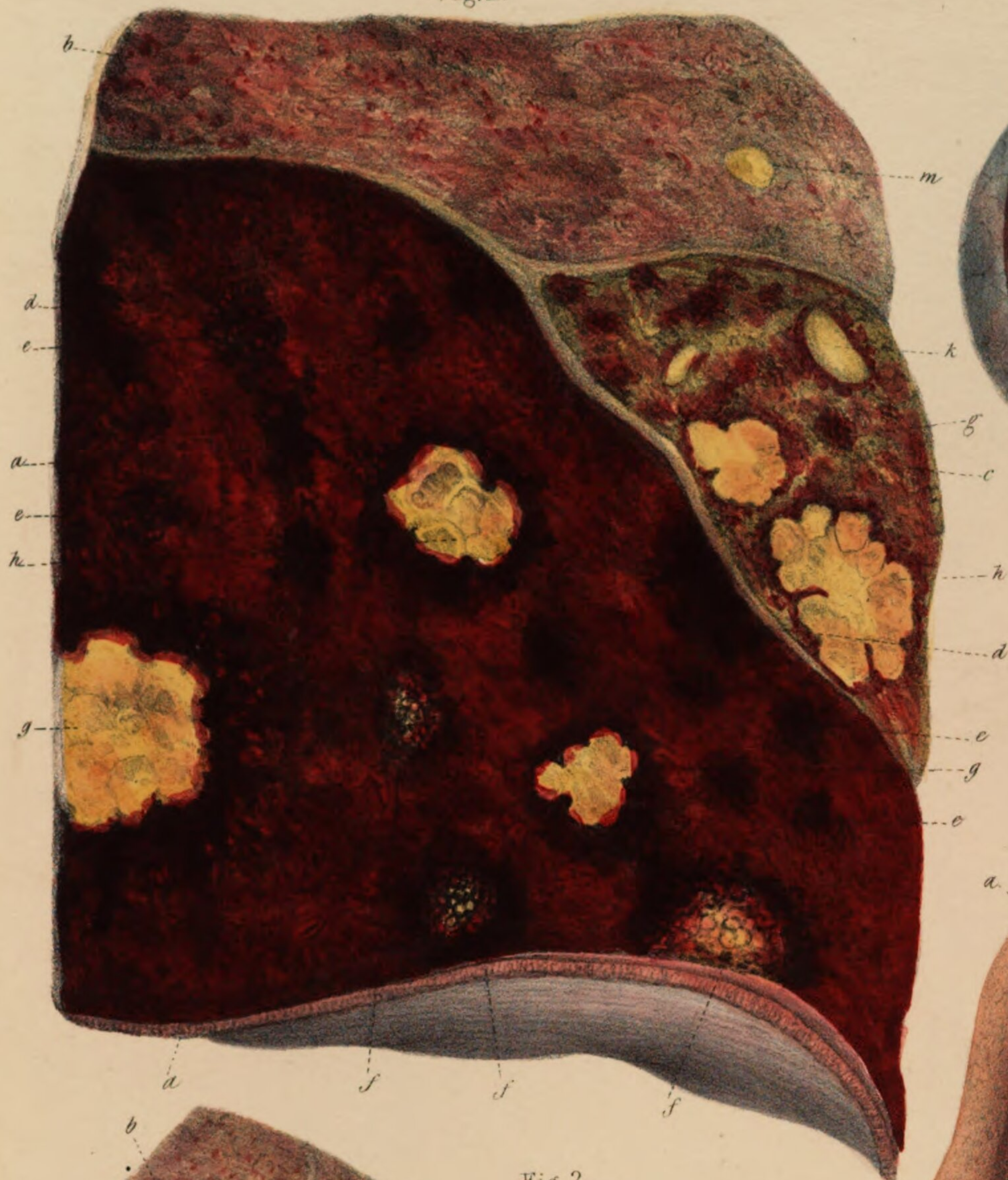


Fig. 2.



Fig. 3.

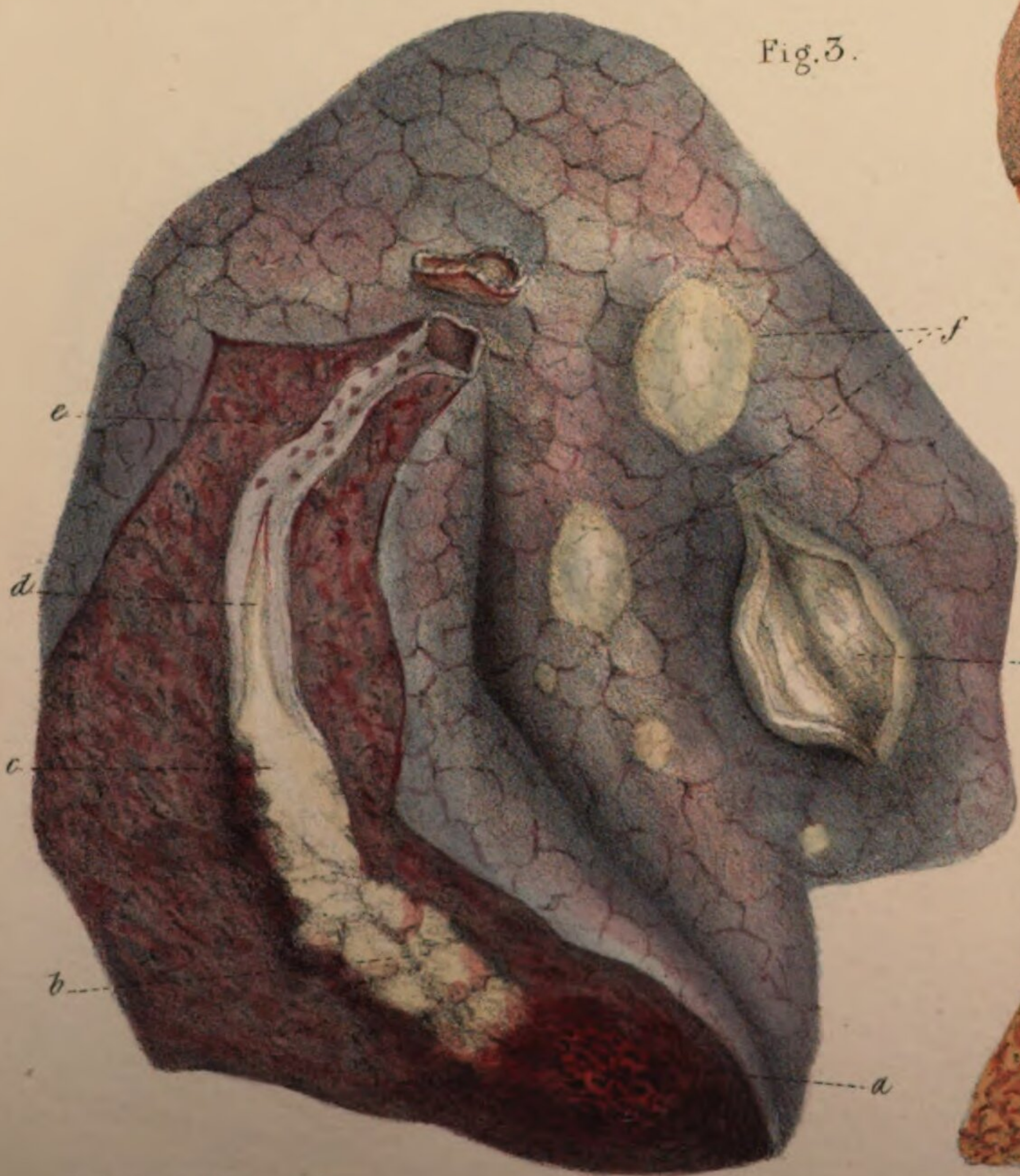


Fig. 4.

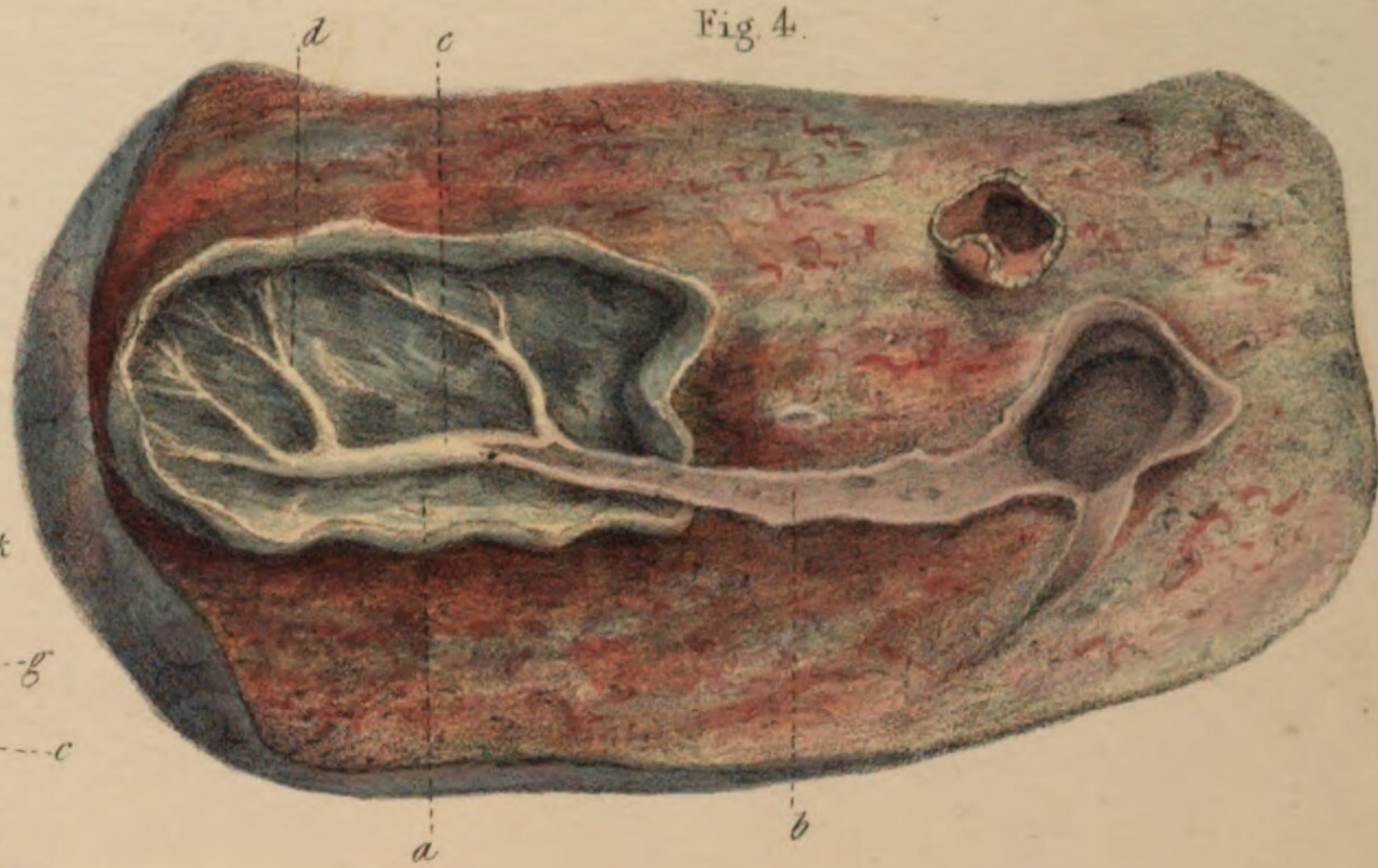


Fig. 5.

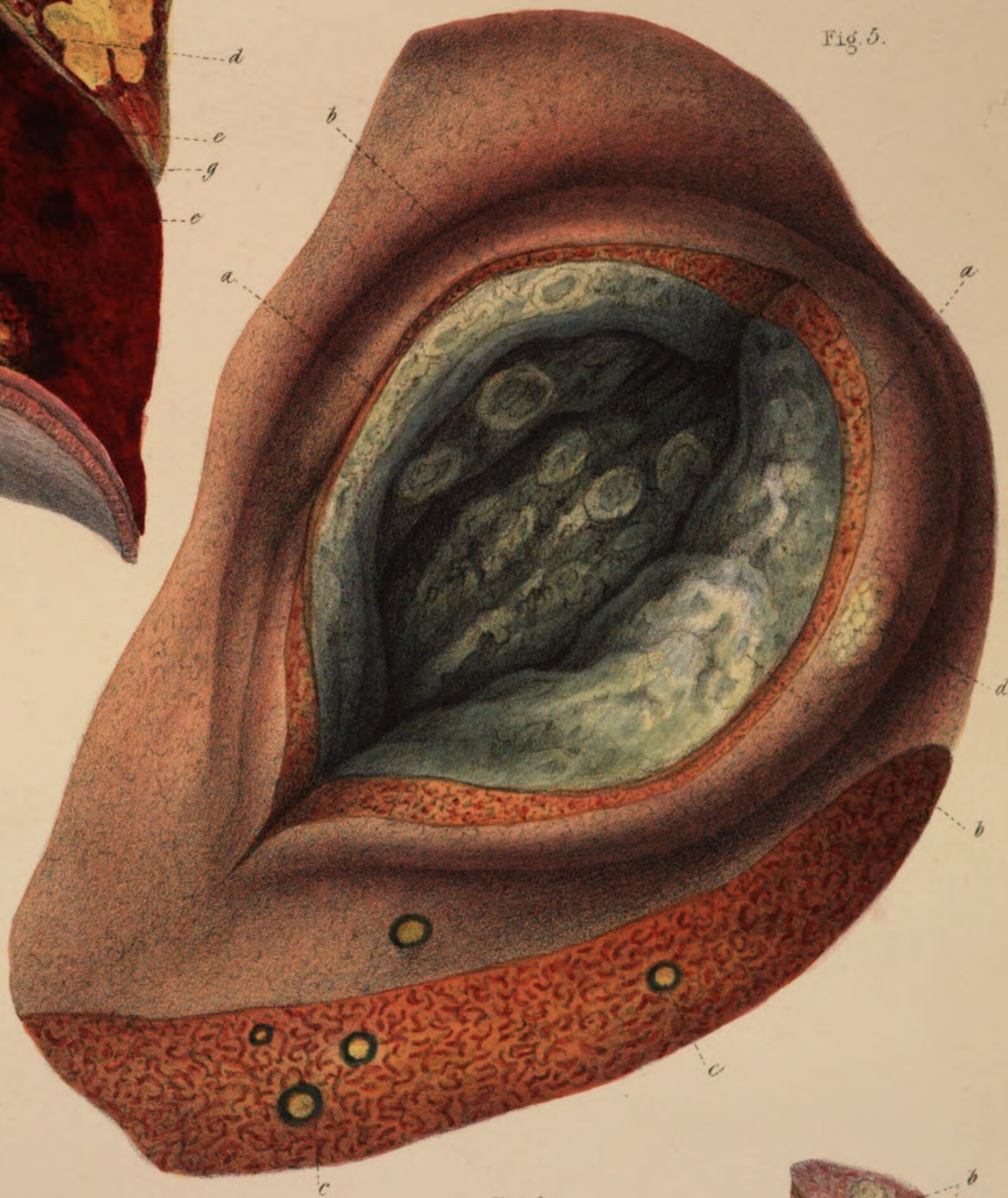


Fig. 6.

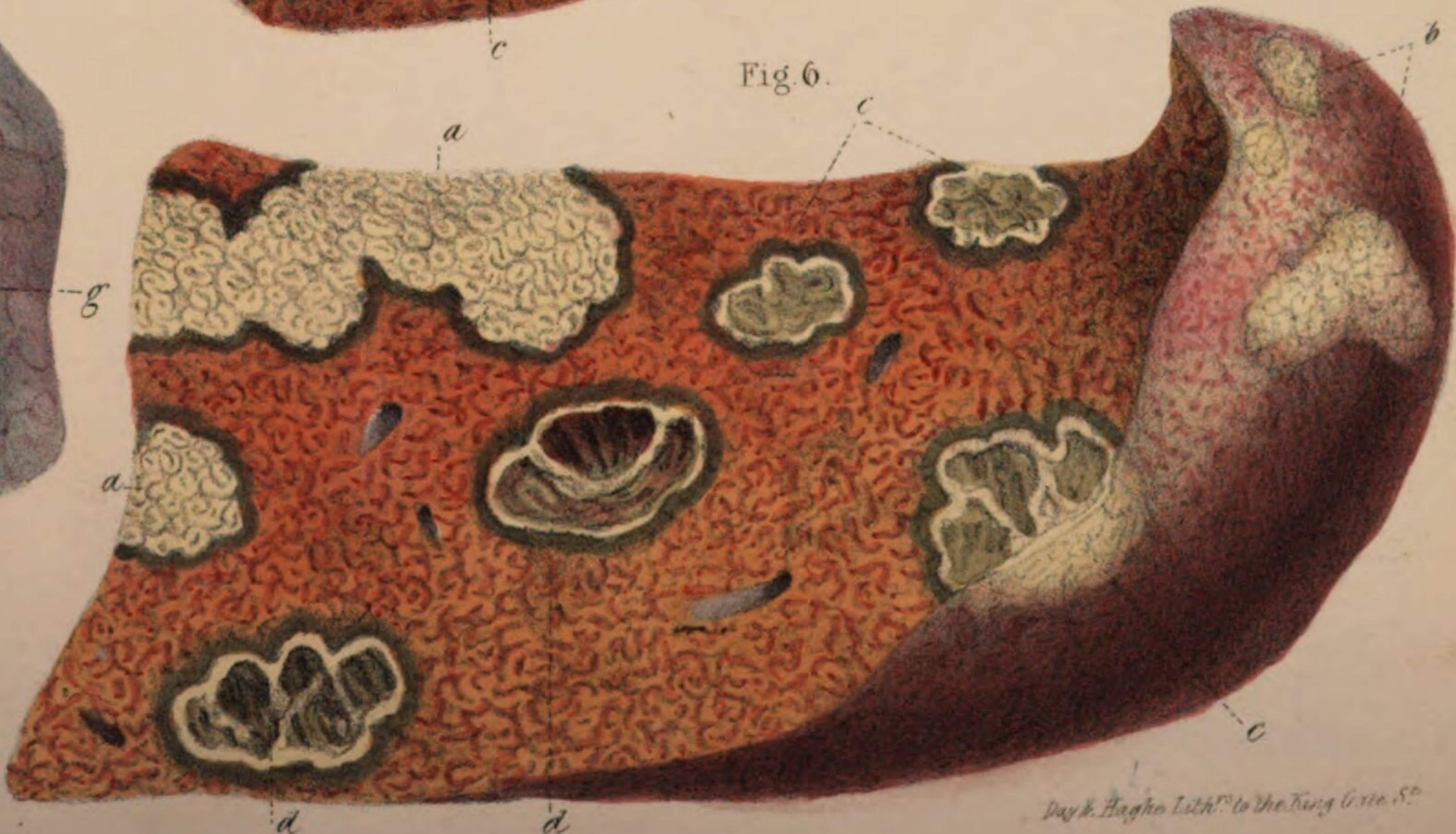


Plate III.

Fig. 3.

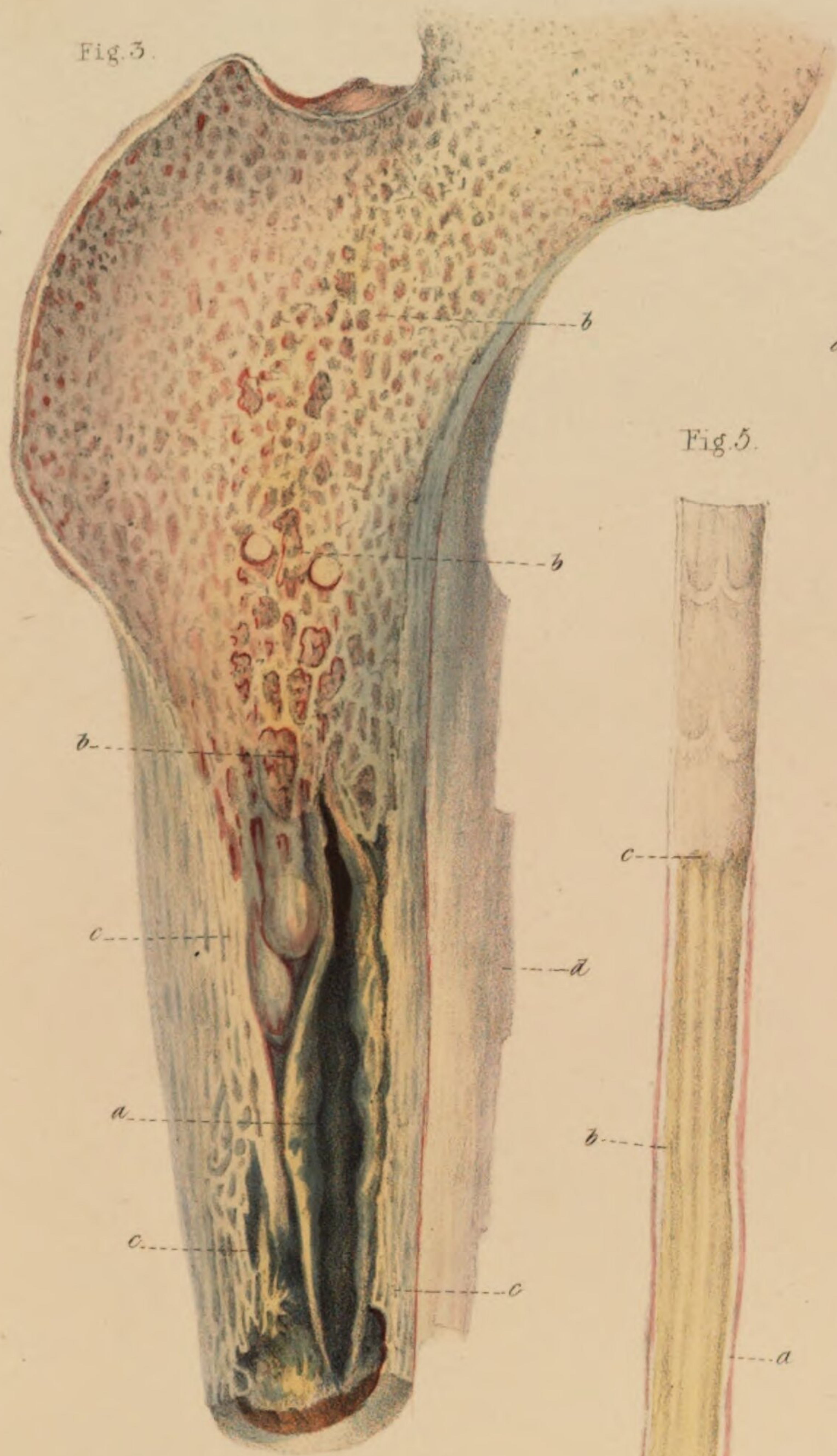


Fig. 5.

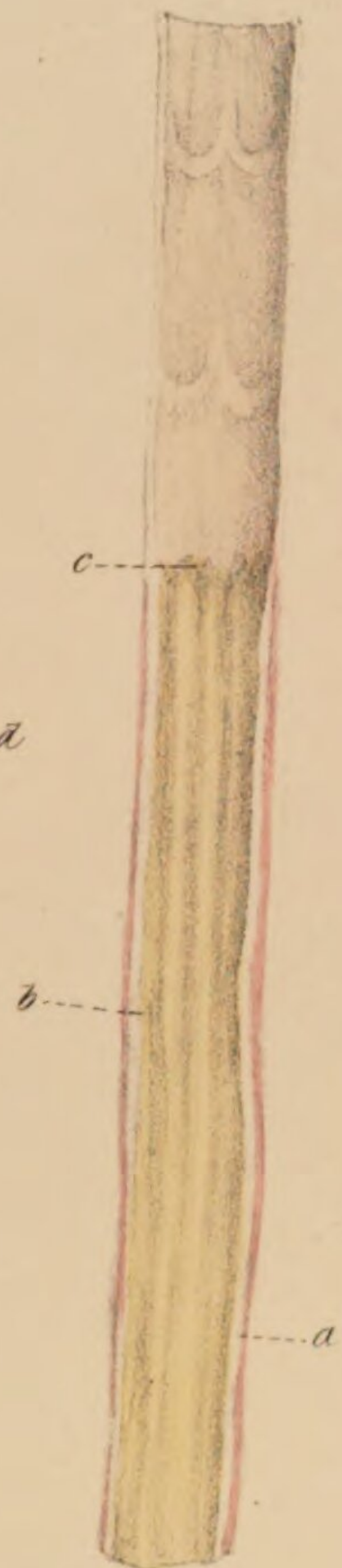


Fig. 2.

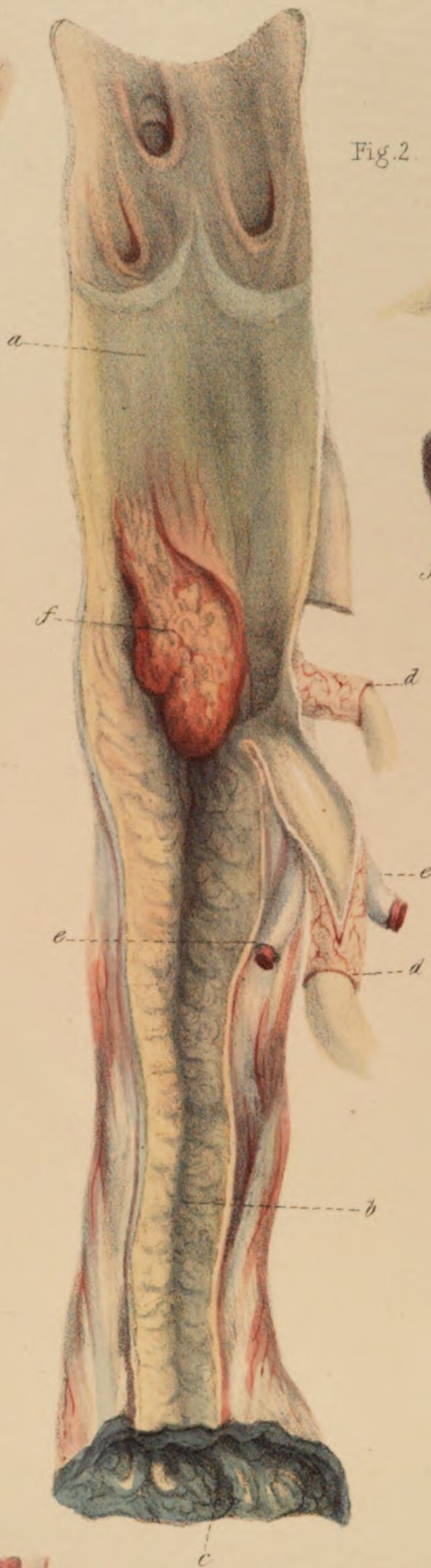


Fig. 1.

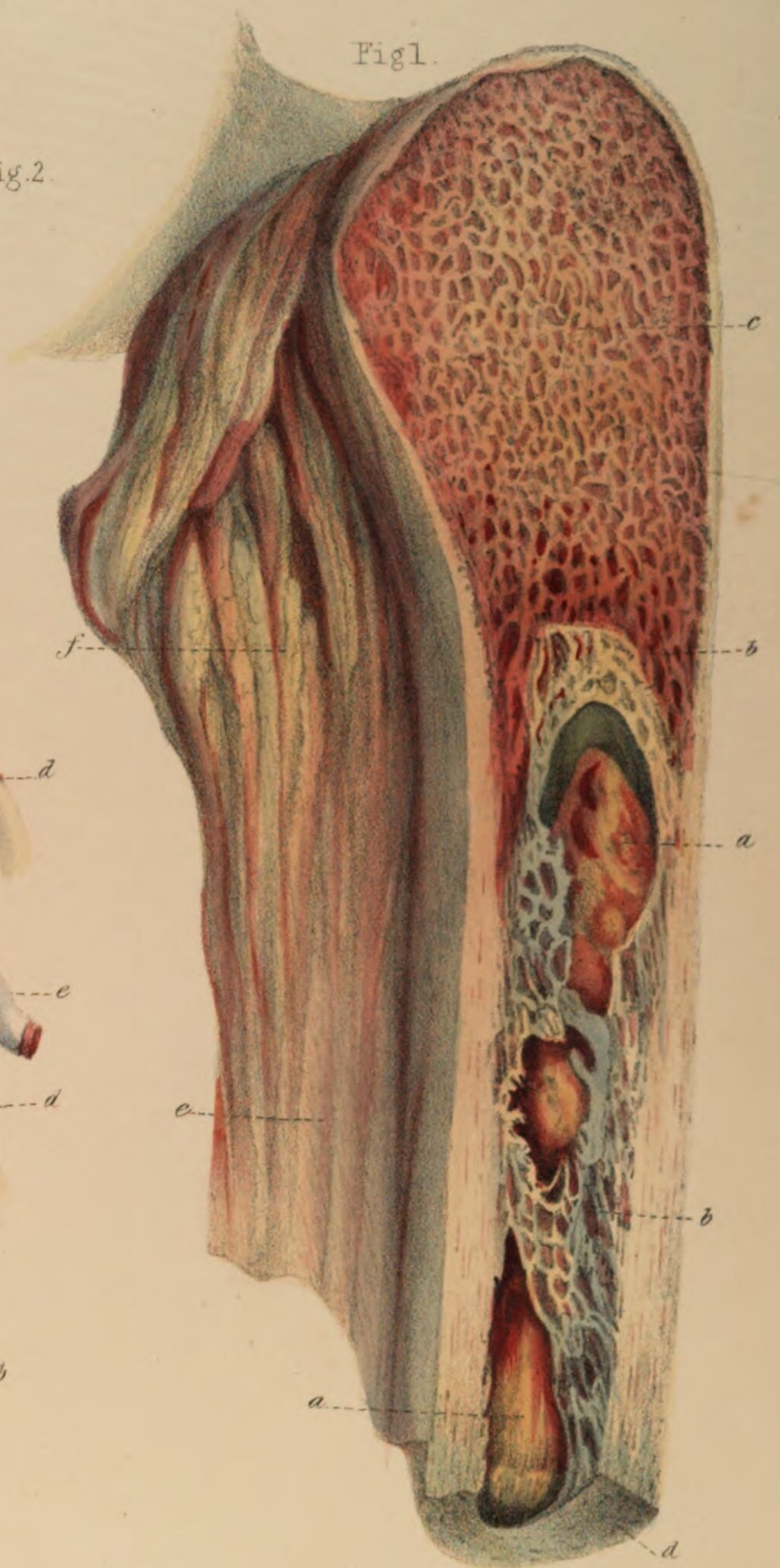


Fig. 4.



Fig. 6.

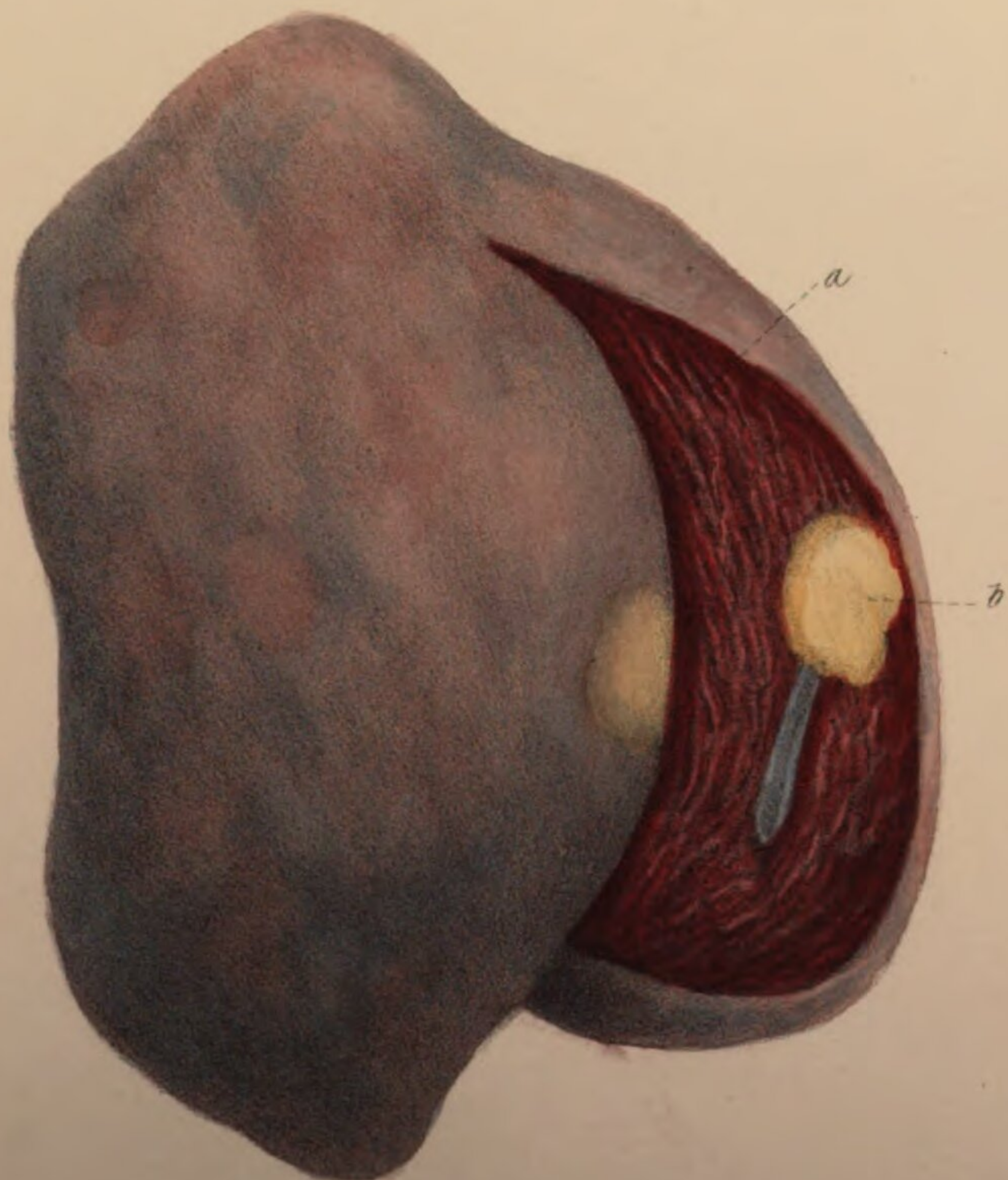


Fig. 4.



Fig. 7.

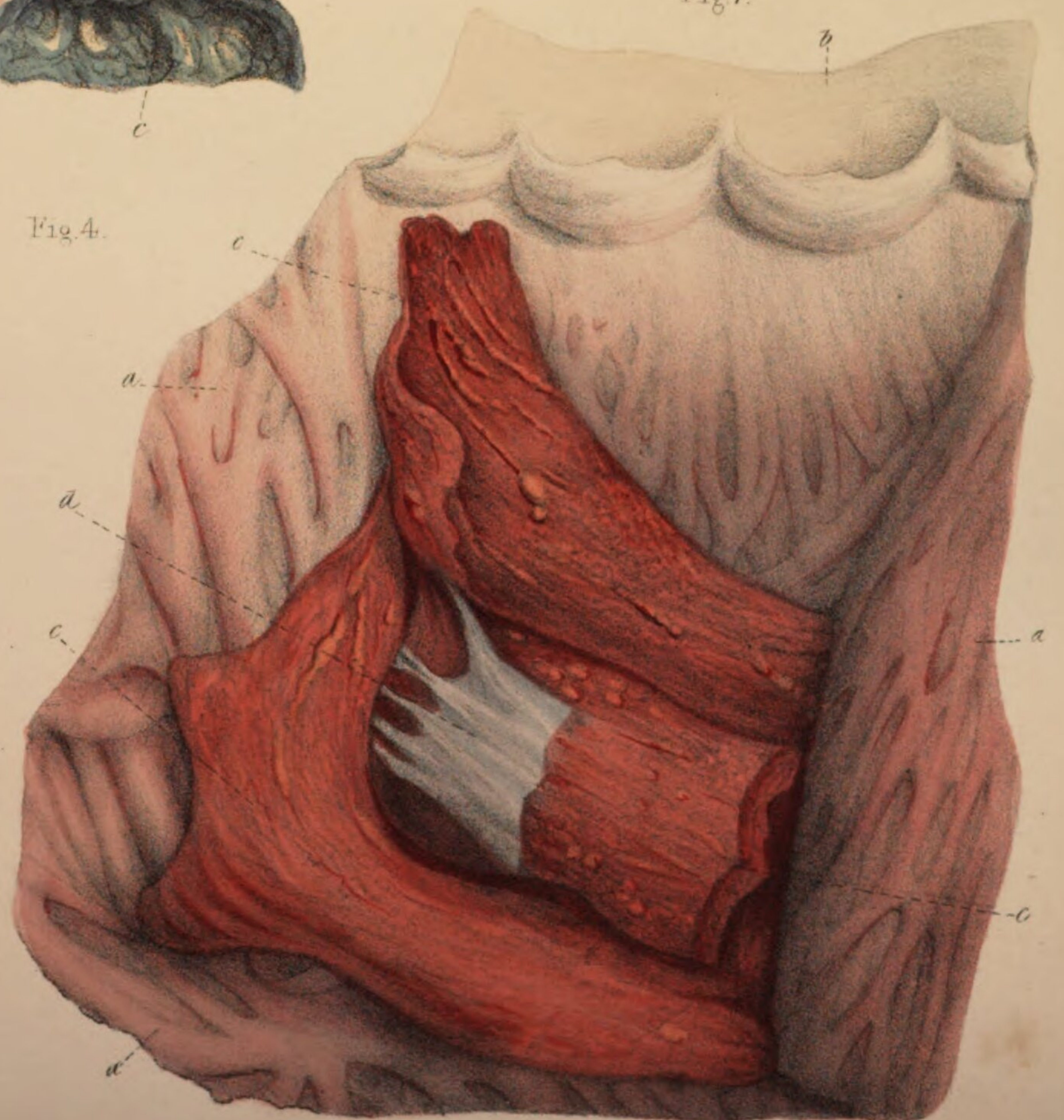


Plate IV.

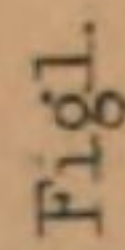


Fig. 2.

R. Curswell ad nat. del.

Day & Haghe, Luth^r to the King.

MORTIFICATION.

THE term mortification is generally employed in this country to express that state which has been induced in a part of the body by the complete and permanent extinction of its vital properties. On the Continent, however, the term *gangrene* is employed to signify the same state, whilst in England it is more commonly used to denote the incipient stage of mortification; a state of a part in which, in the words of the distinguished author of "Lectures on Inflammation," there is a diminution, but not a total destruction of the powers of life; in which the blood appears to circulate through the larger vessels; in which the nerves retain a portion of their sensibility; and in which, perhaps, the part may still be supposed to be capable of recovery: the extinction of the powers of life, the complete cessation of the circulation, and an entire want of sensibility, characterize the second or last stage of mortification, which is called *sphacelus*, whether the dead part has or has not become putrid, whether it has been separated or not from the living parts. Again, some pathologists confine the term *gangrene* to the death of the *superficial* textures of parts; and *sphacelus* to the death of the *whole substance* of an organ, as of the soft parts and bones at the same time.

Other denominations have been given to this pathological state, founded on particular conditions of the affected part, which have been observed to precede or accompany mortification. Thus we have what is called *hot gangrene*, or that form of the disease which is preceded or accompanied by inflammation; and *cold gangrene*, or gangrene without inflammation. We have also *humid gangrene*, from the affected part containing a greater or less quantity of decomposed or other fluids; and *dry gangrene*, when these fluids are not present, or only in very small quantity, and which being frequently the case in gangrene affecting the external parts of the body in old people, has, on that account, been also named *gangræna senilis*.

As the descriptive characters of mortification were originally drawn from the appearances which this disease presents when it attacks the external parts of the body, they have ever since been employed by the pathologist as the means of enabling him to detect it in internal organs after death. It may, however, be fairly questioned whether the application of the term mortification has not been too restricted; and whether parts deprived of their vitality and separated from the living tissues, should not be designated by the same appellation as those which, similarly situated, differ from them only in point of colour and perhaps smell. Softening of the cerebral substance, of the mucous and frequently of the serous membranes, constitutes a state of positive death, but the softened substance, in those instances, presenting neither the peculiar colour nor odour of external parts when mortified, it has been considered proper to distinguish softening from mortification by a term expressive of its principal character—that of softness. I shall, therefore, treat in the present fasciculus only of those diseased states which are usually comprehended under the term mortification.

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Considered in a general point of view, and in relation to the causes by which it is produced, or the morbid conditions of the part in which it occurs, we find that mortification takes place in a variety of ways, and under very different circumstances. A knowledge of these facts suggests the propriety of arranging the several kinds of mortification under the three following heads:—

1. Mortification from cessation of the circulation.
2. Mortification from the violent operation of mechanical, chemical, and physical agents.
3. Mortification from the deleterious influence of certain poisons.

MORTIFICATION FROM CESSATION OF THE CIRCULATION.

Cessation of the circulation in a part of the body may be produced,—1st, by inflammation; 2d, by mechanical causes, which obstruct the passage of the blood; and, 3d, by local or general debility.

I. *Mortification from Inflammation.*—There is no tissue or organ of the body which may not become affected with mortification as the immediate or mediate effect of inflammation. Mortification is, however, much more frequently observed in those organs in which the vascular system predominates, or in which an inordinate accumulation of blood is readily produced, on account of their greater sensibility and their direct exposure to the influence of those causes which give rise to inflammation. Hence the reason why gangrene and sphacelus occur more frequently in the skin and cellular tissue, mucous membranes and lungs, than in the other tissues and organs of the body, as immediate effects of inflammation; and why they are so rarely observed in serous and fibrous tissues, which contain few or no bloodvessels. Not only is mortification rarely observed in these latter tissues, but it may also be said never to occur in them as an immediate effect of inflammation, for they are never found in a state of gangrene or sphacelus, unless the cellular tissue with which they are in contact, and from whose vascular system their nutrition is derived, has previously been diseased. Such also is the case in caries, (death of bone,) as a consequence of inflammation of the periosteum and medullary membrane.

These circumstances enable us to explain why, in many cases, mortification takes place in one tissue and not in another, although the inflammation by which it is preceded is the same in kind, degree, and duration. There are, however, many other circumstances of perhaps still greater importance, the single or conjoint operation of which favours, in a most remarkable manner, the termination of inflammation in gangrene and sphacelus: such as a state of chronic inflammation of a portion of an organ accompanied by induration and obstructed circulation; a state of local congestion, depending on the presence of an obstacle to the return of the venous blood; that state of general debility which prevails at the termination of protracted fevers, or during the first period of convalescence; and a morbid condition of the blood, such, for example, as that which occurs in scorbutus.

General Phenomena of Gangrene and Sphacelus.—As the more remarkable changes which take place in the circulation, innervation, temperature, colour and consistence of a part affected with gangrene or sphacelus, are seldom observed unless on the external surface of the body, we shall describe them as they occur in the skin and subjacent textures.

When inflammation is about to terminate in gangrene, the inflammatory redness assumes a darker tint; it becomes deep purple, livid, or almost black; the temperature

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of the part diminishes, but not always its consistence, which, on the contrary, may be increased, from the presence of accumulated fluids; small vesicles appear on the surface of the skin, formed by the effusion of serosity, or serosity and blood, under the epidermis; the sensibility of the part, which, as well as the temperature, was previously increased, is now much diminished, and the seat of the pain which accompanies the inflammation is transferred to the deeper-seated tissues.

When these modifications of colour, consistence, temperature, and sensibility, continue to increase and terminate in sphacelus, the part thus affected assumes a still deeper tint, or it becomes of a dirty brown or black colour, sometimes grey, greyish yellow, or greenish. The vesicles or phlyctenæ become more numerous and larger, or the whole of the epidermis covering the sphacelated part may be completely separated, raised in the form of a large blister filled with bloody serum, or ruptured and lying in wrinkles on the denuded and discoloured cutis. The skin and cellular tissue beneath the epidermis are swollen and puffy, and crepitate when pressed, or they are soft, flaccid, and cold, and may be cut, pinched, or otherwise stimulated without pain or feeling of any kind being induced; and, lastly, the sphacelated surface emits a strong cadaverous odour. When these latter appearances present themselves, but more particularly when the peculiar odour of gangrene is perceived, they may be regarded as positive signs not only of complete death of the affected part to a certain depth, but also that putrefaction has already taken place. The local emphysema and fetor of putrefaction produced during life, constitute, therefore, signs of great value in mortification. Their absence, however, furnishes no proof that local death may not have taken place; for putrefaction or chemical decomposition of an organ may not follow as a consequence of the cessation of those powers by means of which it was enabled to resist the injurious influence of external agents, until some time has elapsed, the length of which will depend on various circumstances, but more especially on the quantity of fluids contained in the affected organ, and the degree of temperature to which it is exposed.

So long as gangrene continues to spread, the dark colour by which it is characterized is diffuse, and looses itself insensibly in the surrounding skin; but when it is about to terminate favourably, the dark-red colour becomes more circumscribed, gradually disappears, and is replaced by a brighter red, which extends over the affected surface, accompanied by a diminution of the swelling and pain. By-and-bye, the blood having resumed its wonted course, the natural temperature returns, and the healthy characters and functions of the part are restored, without a solution of continuity having taken place.

A similar change of colour is observed to precede the cessation of sphacelus. It appears in the form of a narrow circle, indicating the boundary which separates the living from the dead parts, and announces that adhesive inflammation, the means which nature employs to arrest the progress of the disease, has commenced. Ulceration then takes place along the internal border of the inflamed skin, and a separation is thereby effected between the living and dead tissues, the latter falling off in the form of what is called a *slough*. The loss of substance which is thus occasioned is repaired to a greater or less extent by means of the coagulable lymph which is thrown out on the denuded surface, and which, becoming organized, assumes a membranous or granular form, according to the situation of the part or the nature of the tissue to be repaired, and constituting ultimately what is denominated a *cicatrix*.

Such are the general characters of mortification produced by inflammation of the skin and subjacent textures. In other tissues and organs they are more or less modified;

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the differences which they present depending principally on the degree of vascularity and sensibility of the affected part. Hence the variety observed in the quantity of blood, and the kind and extent of the effusion by which it is followed in inflammatory gangrene and sphacelus; the changes of bulk which organs undergo in either of these stages of mortification; the extent to which their temperature and sensibility may be reduced before they become actually dead, and the length of time that elapses from the commencement to this the ultimate effect of the disease. Examples of some of these varieties will be found in the following details on mortification of particular tissues and organs.

Mortification of particular Tissues from Inflammation.—The *cellular tissue* is not only more frequently the seat of mortification, but it is also more extensively and more rapidly destroyed by it than any other tissue of the body. It is in the *subcutaneous cellular tissue* when affected with erysipelas phlegmonodes, where this disease occurs either idiopathically or succeeds to slight wounds or punctures of the arm in bloodletting, and the fingers in dissection, that the phenomena of gangrene and sphacelus are most conspicuous. The redness of the skin which accompanies or succeeds to the inflammation of the subjacent cellular tissue is variable in degree. When the inflammation is confined to the cellular tissue, and is speedily followed by a copious effusion of serosity, which is generally the case, the skin, instead of being red, presents a pale glossy aspect; but when it affects both the skin and cellular tissue at the same time or in succession, the discoloration of the former differs little, if at all, from that which has been already described as occurring in gangrene and sphacelus of the cutaneous tissue. The arm, if the part affected, is swollen, hard, and the skin distended in proportion to the quantity of serosity or sero-purulent fluid which has been effused throughout the whole of the cellular tissue of the part. Independently of a morbid condition of the economy, and more especially of the blood, indicated by a great deficiency of that plastic property so essential to the process of adhesion,—circumstances which are generally admitted to favour the developement of erysipelas phlegmonodes,—it would not, I believe, be going beyond the evidence of facts, to attribute much of the destructive progress of this disease to the rapid and extensive effusion of serous and sero-purulent fluid by which it is accompanied in its first stage. The increase of bulk and consistence of the affected limb is a consequence of the extensive effusion of these fluids; but the effects produced by their presence in the interior of the limb are much more obvious, and enable us to explain several important circumstances, such as the rapid progress and termination of inflammation of the cellular tissue in gangrene and sphacelus. When we examine a limb in which erysipelas phlegmonodes has gone through its several stages, we find that, in the first stage, the cellular tissue situated most remote from the original seat of the disease, is discoloured by the presence of minute vessels distended with dark blood, and contains a considerable quantity of serosity, which is sometimes nearly limpid. As we proceed nearer to the original seat of the inflammation, the quantity of the effused fluids increases to such a degree that the cellular tissue in which they are contained appears, from the great augmentation which has taken place in its bulk, to form the greater part of the limb. In this state it feels hard, but when pressed between the fingers it is readily broken down into small fragments, from which there oozes out in great abundance a sero-purulent, purulent, and sanguineous fluid. This state of the cellular tissue may be regarded as constituting the second stage of the disease, or a state of gangrene. When we arrive at the original seat of the inflammation, none of the physical characters of this pathological state are to be

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seen. The congestion has disappeared, the blood has been transformed into pus, and the cellular tissue and bloodvessels, converted into a soft spongy substance of various colours, are partly detached in irregular masses, and float in the putrid contents of a ragged excavation. Under these latter circumstances, the muscles, tendons, large bloodvessels, and nerves are often laid bare to a great extent.

Such are the morbid appearances presented by the cellular tissue in fatal cases of erysipelas phlegmonodes, the primary and almost exclusive seat of which is this tissue. They would seem to prove that the rapidly destructive effects of this form of inflammation depend in great measure on the mechanical influence exercised by the effused fluids on the capillary circulation. These fluids must compress the neighbouring veins to a degree that will prevent the return of the blood poured into the capillaries; the function of nutrition must then cease to be accomplished, as is proved by the great diminution of cohesion which is observed to have taken place in the cellular tissue in the second stage of the disease; and as there is no tendency towards the formation of coagulable lymph, by the presence of which alone the inflammatory congestion can be arrested, the state of gangrene which this stage indicates must necessarily terminate in sphacelus. It is indeed well known that free mechanical division of the skin and cellular tissue, especially in the first stage of the disease, is the most effectual means that can be employed to arrest its progress,—a mode of treatment the obvious effect of which is to remove to a greater or less extent the distention of these tissues and the mechanical cause by which it is produced.

It may be remarked here that mortification of the adipose tissue always follows that of the skin and cellular tissue, and in fat persons occasions sometimes the most frightful loss of substance.

Besides the *diffuse* form of gangrene and sphacelus of the subcutaneous cellular tissue, there is also a *circumscribed* form which is observed in furunculus, carbuncle, or anthrax. The great accumulation of blood, and the still greater and rapid effusion of serosity which takes place in these circumscribed acute inflammatory affections, produce a state of extreme induration of the cellular tissue, a greater or less portion of which being thus as if strangulated, dies from want of nutrition, becomes separated from the living parts, and is expelled in the form of a grey or straw-coloured spongy or pulpy mass, through an opening made in the skin by a similar process, by ulceration, or a surgical operation. In cynanche parotidæa, or mumps, the cellular tissue of the salivary glands is very similarly situated as in carbuncle. It is this tissue which is the seat of the inflammation, congestion, and effusion; but being prevented by the unyielding nature of the glandular tissue of the organ from accommodating itself to the increased quantity of the fluids poured into it, it soon sloughs, sometimes before the glandular tissue has undergone any remarkable change of colour or consistence, and even before suppuration has commenced.

When *diffuse* or *circumscribed* inflammation of the *sub-mucous* cellular tissue, terminating in gangrene or sphacelus, occurs, it presents the same general characters as those which have just now been described, but seldom proceeds to any great extent. The *diffuse* form of the disease is rarely observed except in the pharynx and larynx, either as a primary affection, or in connexion with erysipelas phlegmonodes of the arms, face, or neck. In these situations, the sloughing of the submucous tissue is very limited, and always accompanied by a corresponding state of the mucous membrane which covers it. The effusions of albuminous and puriform fluids which take place at the same time, occasion a great increase of bulk, produce dysphagia, great difficulty of

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breathing, or complete asphyxia. Hence the frequently fatal termination of the disease before it has passed into gangrene or sphacelus, and which has sometimes been described under the name of serous, albuminous, and purulent œdema of these parts.

The *circumscribed* form of inflammation of the submucous tissue terminating in gangrene and sphacelus seldom occurs as a primary affection. It follows, in general, inflammation of the mucous membrane, but may afterwards proceed to a considerable extent and occasion sloughing of all the other tissues of the organ in which it occurs, but more especially those of the intestinal tube: such is a frequent cause of intestinal perforation, and the fatal peritonitis by which it is followed.

Gangrene and sphacelus of the *subserous* cellular tissue are seldom observed as a consequence of inflammation; but the former state has more often been met with than the latter, and principally in the sub-peritoneal cellular tissue. The great facility with which this tissue is torn in some cases of acute peritonitis, its pulpy softness in some points, and occasionally a certain degree of fœtor, are circumstances which indicate a near approach to, if not an actual state of, gangrene. In the cavity of the pelvis, and in the iliac and lumbar regions, these appearances are more marked and more easily detected. Not only gangrene but sphacelus of the cellular tissue is occasionally found in these regions in consequence of the extension of inflammation induced more especially by chronic and acute affections of the uterus, rectum, and kidneys.

Although inflammation of the *mucous membranes* is not unfrequently observed to terminate in gangrene and sphacelus, such is by no means so frequently the case as was imagined by the older pathologists. Great congestion, some forms of melanosis, and several other kinds of discoloration of this tissue, produced not only during life but after death, were frequently confounded by them with mortification. The mucous membrane of the throat and intestines is more frequently the seat of this disease than that of any other organ. In the former it is occasionally met with to a limited extent in cynanche tonsillaris and pharyngea, and constitutes the distinctive anatomical character of cynanche maligna or angina gangrenosa. In the latter it follows as a consequence of certain forms of acute enteritis, either when the inflammation affects the mucous tissue itself, its follicular structure, or both at the same time. In either of these situations the mucous and follicular textures are primarily affected, and may be converted into sloughs of considerable extent without the submucous tissue being destroyed. When thus deprived of their vitality, these textures are, at first, of an ash-grey or straw-colour, and may afterwards become brown or black. They are, however, frequently of the colour of the matter with which they are in contact, the fluid part of which is readily imbibed by the soft spongy tissue of the slough. The mucous membrane which surrounds the slough is generally gorged with blood, indicating either a state of great congestion, or gangrene. When, however, the gangrenous inflammation is confined to the glandulæ agminatæ, and when the greater part or the whole of the follicular structure has sloughed, little congestion or inflammatory redness may remain. And, besides, if these glands have previously been the seat of disease, as in tubercular phthisis or chronic enteritis, a slight attack of acute inflammation may be sufficient to destroy their vitality, without any remarkable increase of vascularity or redness remaining in them to indicate the nature of the destructive cause. In this, as well as in many other cases of the same disease in other parts of the body, the state of sphacelus must be determined by the colour, consistence, and smell of the part, taken in connexion with the other collateral negative evidence, afforded by the absence of any other cause capable of producing such a state.

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Gangrenous inflammation of the mucous membrane of the air-passages, of the urinary and genital organs, is not of frequent occurrence. In the former it is rarely met with unless as a consequence of the extension of a similar disease of the pulmonary tissue, or of the internal surface of tubercular excavations, in which latter case the gangrenous and even sphacelated state of the mucous membrane is confined to those which open into the excavations. In the latter it is principally met with as a consequence of the direct operation of mechanical causes, such as calculi contained in the bladder and kidneys, the improper use of instruments in diseases of the bladder and urethra, pressure of the head of the child, &c. The physical characters of gangrene and sphacelus of the mucous membrane of the urinary organs in particular, differ in some respects from those presented by similar states of the respiratory or digestive mucous membrane. Mortification of the mucous membrane of the bladder occurs under two forms, the diffuse and circumscribed. In the former the congestive stage is extreme, and often accompanied by hemorrhage, which gives to the mucous membrane a uniform deep-red colour. Along with this state, dark brown or black patches occupy portions of various extent of the mucous membrane, which, as well as the submucous tissue, is easily torn; and lastly, other portions of this membrane are seen partially detached and converted into a soft spongy substance having a strong gangrenous odour. In the latter, or circumscribed form of mortification of the mucous membrane of the bladder, the state of congestion is also very great, and almost always accompanied by effusion of blood, which, being confined to circumscribed portions of the submucous tissue, produces ecchymoses of various extent. The state of sphacelus is observed to take place in the centre of the ecchymoses, by the mucous membrane being converted into a soft grey or straw-coloured patch. In the mucous membrane of the pelvis of the kidney, where circumscribed gangrene and sphacelus occasionally occur, the congestive stage is marked by great vascularity, without being followed by hemorrhage,—a circumstance which may be attributed to the presence of a much smaller quantity of submucous tissue, and the more intimate union between it and the mucous membrane in this situation than in the bladder.

The uterus is the only other organ in which the physical characters of mortification of the mucous membrane require to be noticed.

In this organ the disease is frequently the direct consequence of acute inflammation of the mucous membrane, occurring immediately or soon after delivery. It may be confined to that portion of the surface of the uterus which gave attachment to the placenta, or it may occupy its whole extent. The congestive and gangrenous stage of the disease is indicated by a dark-red or deep venous colour, and occasionally by a deep blue or dirty black colour, probably produced by the chemical action of gaseous products on the blood. A similar discoloration, sometimes a greenish, or more frequently a dirty yellowish-grey colour, is observed in the stage of sphacelus, and is always accompanied by a strong gangrenous odour. The sphacelated tissues are soft, spongy, easily torn, and are generally covered with a putrilaginous fluid substance. When this substance has been washed away, the surface is rough and irregular, sometimes from the presence of the remains of the placenta, but more frequently from an exudation of fibrinous matter, which is occasionally found to cover the whole surface from the fundus to the os tincæ. The substance of the uterus is sometimes affected in this manner to a considerable depth, but it is more frequently only softened without having undergone any other remarkable change. The presence of pus in the fibrous structure of the

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uterus is seldom observed beyond the gangrened or sphacelated surface, but is frequently met with in the veins and lymphatics.

The few general remarks which have been made on gangrene and sphacelus of the subserous tissue shew that the same morbid states must occur in the *serous membrane* itself; for as the bloodvessels of this membrane are principally, if not entirely, derived from those of the cellular tissue with which it is in immediate contact, any disease capable of arresting the circulation in the vessels of the latter, must be followed by a similar result in those of the former, the portion of which so circumstanced being soon deprived of its vitality. This is, in fact, the mode in which serous membranes become affected with gangrene or sphacelus. It is not from their being loaded with blood under the stimulus of inflammation that they die and are separated, but from the supply of their nutritive fluid being cut off, on account of the morbid condition of the circulation in the cellular tissue just alluded to, or from this tissue having been destroyed by ulceration or sphacelus. It is under these circumstances that sloughing of the serous membranes takes place, which is generally followed by a solution of continuity which establishes a preternatural communication between the serous cavity and that of a neighbouring hollow organ.

The colour of the sphacelated serous membrane is generally of an ash-grey, sometimes ochrey from the presence of bile or blood. It is soft or spongy, and frequently does not present any peculiar smell. Before it separates, the subcellular tissue around it is frequently seen injected with fine red capillaries; occasionally, also, all the tunics of the intestine (when this is the situation of the lesion) are pale, and the accidental opening appears as if it had been made by excision. The external border, however, of the opening is smooth, although irregular, whilst on the internal surface of the intestine it is rough or ragged, or presents other marks of being ulcerated: this is the form of perforation which occurs in chronic ulceration of the *glandulæ agminatæ*.

A dark brown, dark blue, or black colour of the peritoneum, extending over a considerable portion of its surface, has frequently been described by old authors as indicating the presence of mortification of this membrane. It has been shown in the fasciculus on *Melanosia* that these discolorations depend on causes very different in their nature from those which give rise to mortification.

The physical characters of gangrene and sphacelus of the pleura are so similar to those which accompany these states of the peritoneum, that they do not require separate description. By far the most frequent cause of sphacelus of the pleura is the presence of tubercles formed under this membrane. Next in the order of frequency is gangrenous inflammation and superficial abscess of the pulmonary tissue. But under none of these circumstances is the perforation of the pleura which follows, the result of ulceration. It is, as has already been stated, the immediate effect of the cessation of circulation or the destruction of the subcellular tissue, whence the nutrition of this membrane is derived.

Gangrene and sphacelus of *fibrous membranes* in general, and *tendons*, take place in the same manner as serous membranes, that is to say, they are observed to slough only when the cellular tissue surrounding them has previously been destroyed. In like manner also the death of *cartilage* and *bone* is effected by previous disease of the perichondrium, periosteum, or medullary membrane.

Inflammation of the *muscular tissue*, terminating in mortification, seldom occurs to any extent, unless when produced by an external injury, and which affects at the same

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time other neighbouring tissues. The physical characters of the disease in this tissue do not present any modification worthy of particular notice.

Having pointed out what appears to be the most important of the physical characters of mortification of most of the elementary tissues of the body, little remains to be said of those which present themselves when this disease takes place in organs or compound tissues; and as they are in every essential circumstance the same in each of these tissues, it will be sufficient to give a description of them in mortification of the pulmonary tissue, in which this disease more frequently occurs, as the direct effect of inflammation, than in any other compound tissue of the body.

When the *pulmonary tissue* is affected with gangrene, its colour becomes of a deep red, approaching almost to black, whilst its consistence equals that of hepatized lung or liver. When pressed, it breaks down between the fingers, and there oozes out from it blood and a dirty white or greenish fluid of the consistence of milk or treacle, having a very disagreeable odour. When the state of sphacelus is produced, the pulmonary tissue, seen under the pleura, appears sunk beneath the surrounding surface, presents a dirty white, yellowish grey, brown, or greenish colour, and frequently, when extensive, a mottled aspect, in which all these tints are perceived; it feels flaccid and pulpy, and, when cut into, appears as if converted into a putrid sanies, in which shreds of pulmonary tissue and bloodvessels float or lie detached, and which diffuses around the most insupportable odour of sphacelus.

Complete death of the pulmonary tissue may take place in several points of the same lung at the same time, but in such cases it is limited in extent, and is much more frequently the result of a septic agent than of inflammation. It is when it is confined to one point that it has been found to extend so as to occupy the fourth, the half, or even the whole of a lobe. In both cases the sphacelated substance may be limited all round by the adhesive inflammation, or it may be confounded with a gangrenous state of the neighbouring tissues. In the first case we have what is called *circumscribed*, in the second *uncircumscribed* mortification. The more extensive, however, the state of sphacelus, the more rarely do we find adhesive inflammation to have taken place.

In both forms of sphacelus, and whether a separation has been effected or not between the living and dead parts, a certain number of the bronchi are laid open, and if the patient survives for some time, the broken-down tissues, converted into a soft dirty pulp, in which sometimes small portions of the pulmonary tissue can be recognised, are expectorated. Occasionally, and when the sphacelus extends to the surface of the lung, the pleura is also perforated.

In some cases in which the sphacelus has been arrested by the adhesive inflammation, portions of pulmonary tissue of various sizes have been found either loose, or attached by obliterated bloodvessels, and floating in the fluid contents of the excavation. In such cases the surrounding pulmonary tissue is consolidated by the presence of organized lymph, and is sometimes covered by a membrane of new formation, which encloses the dead and putrid pulmonary tissue which had not been expectorated. The cicatrization of an excavation of this kind has not, however, so far as I am aware, been observed. On the contrary, excavations of an ulcerous character are formed, the origin of which was first pointed out by Laennec.

State of the Vascular System in Mortification.—When the transparent part of an animal, such as the web of the frog's foot, or the mesentery of the rabbit, is placed under the microscope, and submitted to the stimulus of a mechanical or chemical agent, the capillary vessels of the part, as well as the blood which under these circum-

[*Mortification*, 2.]

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stances flows into them, are seen to undergo a regular series of changes, referable to the hydraulic and dynamic conditions of both, and which constitute the pathological state of a part, called *inflammation*. Without entering into the details which belong to another section of our subject, it will be sufficient for our present purpose to state that when inflammation has arrived at what Kaltenbrunner calls its *perfect state*, that is to say, when the capillaries are distended with blood which has ceased to circulate, the part from a deep red soon changes to a dark brown or black colour. Cessation of the circulation, coagulation, and discoloration of the blood, are the successive changes which announce that the functions of the inflamed part are about to cease. The change of colour which takes place is found to depend chiefly on a corresponding and similar change induced in the blood contained in the vessels of the affected part, or that has been effused during the violence of the inflammatory excitement. Soon after the stagnation of the blood, the globules of this fluid are seen to unite, adhere to the internal surface of the vessels, and form a solid dark-coloured mass occupying their whole caliber. The sensibility of the part rapidly diminishes after the coagulation of the blood, although the nerves themselves are not observed to undergo any perceptible change. The coagulation of the blood is also followed by the cessation of absorption, for the most active poisons introduced into a part the vessels of which are thus obliterated, either produce none of the effects which are peculiar to them, or do so very tardily and ineffectually, in which latter case they may have found their way beyond the obliterated vessels by imbibition. Under these modifications of function, nutrition, after a certain length of time, ceases in the affected part, the temperature of which also sinks to that of external objects. Its consistence diminishes so soon as decomposition commences, and the colour which it assumes varies with the quantity and quality of the fluids which it contains, and the chemical changes which these undergo from the action of the gaseous products of decomposition.

The state of the vessels and of the blood which precedes the physical signs of mortification may be regarded as representing that state of a part which we call gangrene. The blood has ceased to circulate, it is even coagulated: the application of artificial stimuli to the neighbouring tissues furnishes no evidence of their possessing sensibility or contractility. Yet, as under these circumstances we know that actual death may not have taken place, that the blood may resume its fluidity and circulate anew, and sensibility and contractility again return, the state to which I have just alluded may properly be regarded as furnishing us with what may be called the anatomical and physiological characters of gangrene,—a state in which the functions of a part are suspended, analogous to that of the whole body in asphyxia, whether we regarded it as susceptible of recovery, or as an intermediate state which separates inflammation from mortification.

When recovery from the state of gangrene is about to commence, circulation becomes more active all round the circumference of the affected part; the coagulated blood gradually disappears by the separation of its globules, and their transmission into the neighbouring currents; absorption is manifested by the more or less rapid removal of the effused fluids, sensation and motion return, and the part is restored to the healthy state.

The changes which mark the transition from gangrene to sphacelus have already been enumerated. The separation of the dead part takes place within limits fixed by the state of the vascular system. Thus, the line of demarcation between the dead and living parts does not extend beyond the obliterated vessels; for before adhesive inflammation has taken place, they are found obliterated to a greater or less distance beyond the mor-

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tified part. Their obliteration is not necessarily the consequence of the effusion of coagulable lymph. It depends on the plastic property of the blood contained within the vessels, which unites with their lining membrane, becomes organized, and thus secures their permanent obliteration.

It is also to the obliteration of the bloodvessels alone that immunity from one of the most dangerous consequences of mortification, viz. hemorrhage, is to be ascribed. The presence of coagulable lymph, its organization and union with the parts into which it has been effused, constituting what is called adhesive inflammation, contributes no doubt to prevent hemorrhage during the process of separation of the dead part, or sloughing. But I am disposed to believe that it is the prevention of hemorrhage from the smaller vessels only that is secured by the adhesive inflammation, while that from the larger ones is prevented by the previous coagulation of the blood within them. That it is to the coagulation of the blood in the large vessels of a limb that we must attribute the non-occurrence of hemorrhage after sloughing, is rendered still more evident from what occurs in some cases of extensive and spreading gangrene, of the inferior extremity for example, and to arrest which it is found necessary to have recourse to amputation. The limb is removed, but the large bloodvessels yield little or no blood; they are, in fact, obliterated by firm coagula. There is no adhesive inflammation in such cases, and gangrene and sphacelus succeed to the operation, in consequence of the vessels not having been divided above the point at which they were obliterated.

Such is the state of the bloodvessels which I have found to accompany sphacelus without hemorrhage. When, on the contrary, hemorrhage occurs in this stage of mortification, these vessels are pervious and filled with fluid or imperfectly coagulated blood; and the cellular and other tissues are more or less infiltrated with serosity, sero-sanguinolent, and puriform fluids.

II. *Mortification from a mechanical obstacle to the Circulation of the Blood.*—The blood may be prevented from arriving at, or returning from, a part of the body by mere mechanical causes. In both cases mortification is the consequence of the cessation of the function of nutrition, either from a deficiency of the arterial or the stagnation of the venous blood. A deficiency of the arterial blood may be occasioned by ligature of the principal artery of a limb; by coagulated blood; organized or unorganized fibrine, occupying the entire caliber of such an artery or its larger branches; by ossification of the walls of these vessels, or their conversion into a solid fibrous or ligamentous tissue.

Stagnation of the venous blood may depend on obliteration of the veins caused by the pressure of tumours; by accidental products formed in their cellular sheath; by the presence of fibrine or other solid substances derived therefrom, formed within the veins, and either simply lodged within them, or more or less intimately connected with their lining membrane; and, lastly, by diseases of the heart, which greatly obstruct or prevent the return of the venous blood to this organ.

Although the physical characters of mortification produced by a mechanical obstacle to the *venous circulation* present considerable variety in different organs, they present one common character, viz. an excessive accumulation of blood in the venous system: trunks, branches, and capillaries of the affected part. It is necessary, however, to observe that the presence of this state of the venous system, as well as the degree and extent to which it exists, is not always obvious, and for reasons which will be pointed out presently.

The physical characters of the present form of mortification when succeeding to disease of the heart, are no where so conspicuous as in the inferior extremities, and may be regarded as furnishing us with an accurate view of those which accompany the same

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disease when produced in a similar manner in internal organs. The first local sign that an obstacle exists to the return of the venous blood from the inferior extremities, is manifested by the appearance of slight œdema around the ankles. The serosity gradually accumulates in those parts, spreads from thence throughout the cellular tissue beneath the skin and between the muscles; the feet, and afterwards the legs, thighs, &c. become swollen; the skin assumes a smooth and glossy aspect, feels tense, and sinks into the cellular tissue when pressed, and does not resume its former shape and situation till raised by the return of the serosity beneath it. The colour of the skin, at first natural, becomes pale and waxy, and may continue in this state during the greater part of the course of the disease. When discoloration of the skin is about to take place, it is seen to depend on the presence of subcutaneous veins, which gradually increase in bulk and number, coalesce in several points, and communicate a slightly mottled aspect to the skin, of a dull red or purple colour. On one or more of these points where the congestion is greatest, and where the skin is less yielding, as over the tibia and above the malleoli, phlyctenæ or large bullæ are formed by the effusion of serosity, either alone or mixed with blood, under the cuticle. When these burst, the cutis beneath presents a dark red or brown colour, and very soon is converted into a dirty yellow or ash-grey slough. The separation of the slough is sometimes preceded by an increase of redness in the surrounding cutis, which, from its anatomical characters, and the increase of temperature and pain by which it is accompanied, is obviously of an inflammatory nature. At other times the redness which precedes or accompanies the separation of the dead part is very slight, and is evidently owing to mere venous congestion, occasioned not only by the disease of the heart, but also by the serosity accumulated in the cellular tissue of the limb, which, from the pressure which it occasions, further retards the return of the blood, and aggravates all the symptoms of the disease. It is, indeed, to this secondary obstacle to the return of the venous blood of the limb, that the termination of the disease in mortification is chiefly to be attributed. It is likewise in consequence of the accumulation of serosity beneath the skin that the state of congestion of the venous system of the limb is not at first perceived.

Although, after the separation of the slough, a loss of substance of considerable extent in depth may appear to have been produced, it perhaps never proceeds beyond the cellular tissue; and it is in consequence of this tissue being greatly distended with serosity, that the loss of substance which follows sloughing appears to have penetrated deeply into the substance of the limb. Congestion, gangrene, and sphacelus may take place on several parts of the leg, but they are in general limited to the parts which have been noticed, and rarely occur on the feet or toes.

The separation of the sloughs is seldom followed by hemorrhage; and when it does occur, it consists merely in a slight oozing of blood from some points of the denuded surface. This circumstance, as well as the state of the blood generally found in the veins after death, is readily accounted for. The blood in the veins in the immediate vicinity of the sphacelated part of the limb is found coagulated, or these vessels are filled with fibrine. More remote from this part, and sometimes throughout the greater extent of the limb, the blood with which they are distended is more or less coagulated, but becomes more fluid as we examine it farther from the seat of the sphacelus.

As it would not be of much practical importance to enter into a description of the physical characters of mortification of internal organs, from a mechanical obstacle to the return of the venous blood, I shall only briefly notice some of those diseases which occasionally give rise to this state of the circulation. Mortification of internal organs from this

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cause is always limited in extent, and is seldom observed except in the lungs, liver, or intestines. It occurs in the lungs when their cellular structure has become consolidated by the slow deposition of coagulable lymph, and has produced that state termed hepatisation. The pulmonary tissue in this state may be as firm as liver or as hard as cartilage; sometimes grey, purple, livid, or nearly black. When carefully dissected, not only are the veins contained within it found compressed or obliterated, but also the arteries are much diminished in bulk. Such is sometimes the state of the vessels in the indurated walls of tubercular excavations, as well as of those which traverse the septa. A similar state of the bloodvessels, and induration of the pulmonary tissue, may likewise be produced by an extensive deposition of tuberculous matter. Under such circumstances, portions of the indurated pulmonary tissue become deprived of their vitality, and are separated without the supervention of inflammation.

Sphacelus of portions of the liver is not very rare in those cases in which this organ is nearly filled with carcinomatous tumours. These tumours produce, mechanically, extensive obliteration of the veins; and when a portion of the liver becomes incarcerated by them, it is converted into a dark brown slough, generally soaked with blood, and sometimes mixed with softened and detached pieces of the neighbouring tumours.

Mortification from a mechanical obstacle to the return of the venous blood, is well exemplified in intus-susception of the intestines. When the superior portion of intestine passes into the inferior, it carries along with it that part of the mesentery to which it is attached. If it does not suffer much compression, the invaginating process may go on to a great extent; but if it is compressed to such a degree that the return of the venous blood is obstructed, this stage of the disease is arrested, on account of the congestion of all the tunics of the invaginated portion. The congestion is not the consequence of inflammation; it is produced by compression, and in the following manner:—when the mesentery is put on the stretch by the descent of the superior into the inferior portion of the intestine, the veins belonging to it are compressed between the walls of both portions, just at the point where the invagination terminates superiorly. If adhesive inflammation takes place at this point, the peritoneal surfaces of both portions become united, and the veins obliterated. As the arteries are much less affected by pressure than the veins, they continue to pour in their blood into the invaginated portion; this fluid accumulates, and produces an extreme degree of congestion of the mucous and submucous coats, giving to them a deep red or almost black colour. In this state, however, the intestine is not deprived of its vitality. It is in a state of gangrene, but not of sphacelus; for its structure is still entire, and it may, when separated and evacuated, present, after having been macerated for some time so as to deprive it of the blood which it contains, the most perfect state of integrity of all its tunics. Occasionally, however, a portion of the whole of the invaginated intestine is found in a state of complete sphacelus, and is passed in the form of irregular spongy masses or shreds of a dirty ash-grey, brown, or black colour.

There are two forms of mortification produced by the cessation of the *arterial circulation*, the first depending on rupture of the internal and middle coats of a large artery, the second on the presence of accidental products which produce obliteration both of the trunks and branches of the arteries of a limb.

Professor Turner was the first who described the spontaneous rupture of the internal and middle coats of an artery, in an interesting paper published in the third volume of the Edinburgh Medico-Chirurgical Society's Transactions. In one of the cases only, however, the histories of which he relates, did mortification take place. The popliteal

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artery was the seat of the lesion, the internal and middle coats of which were found lacerated, and thickened, and its cavity obliterated in several points by coagulated blood, fibrine, and lymph. We are ignorant of the pathological condition of the artery which precedes the rupture of its coats; but the only circumstance which is sought to be established here, is the production of mortification from a cause acting mechanically, and obstructing or entirely interrupting the passage of the blood through the principal artery of a limb.

The second form of obliteration of the arteries which gives rise to mortification, consists, as has been stated, in the presence of fibrine, fibrous or osseous substances formed in these vessels. When the quantity of these substances is such as to interrupt the circulation of the blood through the principal arterial trunk or branches of one of the inferior extremities, mortification is almost always the consequence, from the advanced period of life at which this form of the disease generally occurs, and the very unfavourable state of the arteries to the formation of a collateral circulation. It is to this form of mortification that the term *gangrena senilis*, the *idiopathic* and *dry* gangrene of authors, if employed at all, should be applied.

The first change which announces the occurrence of local death from obliteration of the arteries of one of the inferior extremities, occasioned by the presence of these accidental products, is a dark-red, purple, or almost black discolouration of the fleshy or under-portion of one or more of the toes of the foot. There is, in general, no previous swelling of the affected toes, no increase of their temperature or sensibility. The discoloration alone is often the first circumstance which attracts the attention of the patient to the existence of the disease. In some cases, however, a prickling or tingling sensation, or a certain degree of numbness and cold, are experienced in one or more of the toes, and which, when examined, are already found to be discoloured; not red, hot, swollen, and painful, but of a purple or livid colour, colder than natural, not painful when pressed, and shrunk rather than increased in bulk. The discoloration extends slowly until it has pervaded the whole of the skin covering the toes, then proceeds upwards over the back and sides of the foot, and sometimes mounts nearly as far as the knee, although more frequently death takes place from the constitutional disturbance which ensues, before it has passed the foot or ankle-joint. During its progress, the discoloration generally presents the same purple or livid tint which it did at the commencement, and although it may be preceded by some swelling and congestion of the skin and subcellular tissue, its progress is seldom marked by the bright red colour of inflammation; and when inflammatory redness of the skin takes place, accompanied with heat, pain, and tumefaction, these phenomena must be regarded as effects of the disease which more frequently tend to increase than to interrupt or arrest its progress.

The bulk of the affected parts depends chiefly on the situation and extent of the obstacle to the circulation. If the obstacle be extensive, the quantity of blood admitted to the foot is too small to give rise to congestion; and this not taking place, there is little or no effusion of serosity. Hence there is no increase of bulk in mortification from this cause; and if the obstruction has been effected slowly, the foot and leg may even be atrophied previously to their being attacked with mortification, the dead parts being shrunk, dry, and indurated. These physical characters of the disease are entirely owing to the hydraulic conditions of the limb just alluded to; for if the obstruction to the passage of the arterial blood be only partial, and particularly if it has occurred suddenly, a considerable degree of congestion is induced, and consequently the effusion

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of a greater or less quantity of serosity, whereby the bulk of the foot, and more frequently of the leg, is increased. Even in this case, however, there is not any marked increase of bulk in the toes, the primary seat of the disease. It is in its progress upwards that the congestion and œdema become manifest; that the skin becomes tense and painful; and that the febrile symptoms, if they have not yet occurred, appear, increase rapidly in severity, aggravate the local affection, and hasten its fatal termination.

In every case of gangrena senilis which I have examined after death, the arteries of the limb were obliterated to such an extent as to interrupt the circulation of the blood. The obstructing cause consisted, in five or six cases, of a fibrous tissue formed either in the walls or cavities of the arteries, and which had converted these vessels into nearly solid cords of ligamentous consistence. This state was traced from the toes more than half way up the leg; it was always connected with ossification of the larger branches and trunks of the thigh and other parts of the body. In other two cases, the obstruction depended on extensive ossification of the principal arteries of the limb; and in several others it was produced by solid fibrine formed around spicula of bone projecting from the internal surface of the arteries. Connecting these states of the arteries with the external appearances of the mortification with which they are accompanied, there cannot, I conceive, remain a doubt that this form of the disease is the immediate consequence of a deficient supply of arterial blood, from a mere mechanical obstacle to the circulation of this fluid. So palpable indeed, and so frequent are the morbid conditions of the arteries which I have described in gangrena senilis, that it is more than surprising that any individual who has had opportunities of investigating this disease should have attempted to ascribe its origin to inflammation of these vessels. Leaving aside the incontrovertible evidence of the material facts which demonstrate the truth of the position which I have laid down, there are other circumstances which show that inflammation of the arteries cannot be the cause of gangrena senilis such as I have described it. Whether the inflammation which is supposed to give rise to the disease be considered as of an idiopathic or symptomatic kind, is of no import in the decision of the question. For, in the first place, the obstructing cause, viz. fibrous, fibro-cartilaginous, and osseous tissue, could not owe its origin to inflammation in a space of time so short as that which often marks the duration of the disease; and in the second place, the presence of these accidental tissues in the arteries is no proof that inflammation had ever existed in these vessels. Stagnation of the blood from mechanical or physical causes is sufficient to give rise to the formation of these tissues by means of the fibrine of this fluid.

But it is maintained that the disease to which the ambiguous and improper term of gangrena senilis has been applied, is always symptomatic of acute inflammation of the arteries. It is not, I believe, going too far to assert that we have as yet no evidence of idiopathic inflammation of the large arteries giving rise to mortification. The opinions and observations which have been laid before the public on this subject, in the "*Leçons Orales*" of Mons. Dupuytren, are equally inconclusive and defective. The appearances described in that work as evidences of inflammation of the arteries giving rise to mortification, might, with greater propriety, be considered as characterizing some of the worst forms of phlebitis, the disease which, in my opinion, actually existed in the woman whose case is detailed in the First Observation.

III. *Mortification from debility.*—We have seen that mortification of various parts of the body may be produced by mechanical causes whose operation is entirely limited

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to the vascular system, in which they impede or arrest the circulation of the blood. This fluid, as well as the solids in general, may be in the healthy state up to the moment at which the local mechanical cause begins to operate and manifest its effects; and these are characterized by a diminution of all those properties the extinction of which is the death or mortification of the part thus circumstanced. A similar healthy condition of the solids and fluids may precede mortification from inflammation, but the phenomena of the disease are the opposite of the former. Local death does not take place until after the morbid stimulus has increased the vital properties of the part to their maximum. The part is thus exhausted of its strength, if we may be allowed the expression, and being deprived also of the means of renewing it, from the changes which have taken place in its more essential elements, it soon sinks into a state of absolute death. In the form of mortification which we have now to consider, there is a previous state of disease,—a state of local and general debility, and which constitutes its essential character. The physiological and physical properties of the fluids and solids of the body are so modified, that every function of the economy is slowly, ineffectually, or imperfectly performed. Innervation and nutrition in particular are so circumstanced, that even those agents on which the varied phenomena of health and life more or less immediately depend, now become the causes of disease and of death. Notwithstanding that this form of mortification differs so much in these respects from the two preceding forms, it has appeared to me that they may be classed with propriety under the same general head, inasmuch as one common condition of the affected part, viz. cessation of the circulation, is the immediate cause of local death in all of them.

In mortification from debility, a local accumulation of blood constitutes, in general, the first perceptible change in the part which is about to be deprived of its vitality. This may take place from the part being submitted to pressure merely from its own weight or that of the body, from slight friction, puncture, or other similar causes. In some of these cases the blood accumulates, partly from the influence of gravitation, and partly from compression of the veins; as, for example, in mortification of the soft parts covering the sacrum, heels, elbows, &c. of persons recovering from typhoid fevers, and who are left in that state of prostration which precludes the possibility of changing the position of the body. It is, perhaps, still more conspicuous in some patients similarly confined with paraplegia from injury of the spinal cord.

A state of local congestion is also frequently the only change which precedes the sphacelus of the skin to which leeches have been applied, or which has been scarified or punctured, in greatly debilitated scrofulous children. The skin around the leech-bites assumes a dirty purple, livid, or almost black colour; looks sometimes as if it had been injected with ink; presents no previous redness, heat, or pain, and is not swollen except where the blood is accumulated; it drops off in the course sometimes of twenty-four hours, leaving a number of circular openings, which unite and spread by similar succeeding congestions and sloughing of the contiguous skin.

The occurrence of mortification in scorbutus affords another striking example of the influence of general debility in the production of this disease. Portions of the skin often become gorged with blood, die, and slough, without our being able to discover that they have received any previous injury.

There is one other form of mortification from debility which occurs generally in children, and has received various appellations, such as, *noma*; *stomacace gangrenosa seu maligna*; *necrosis infantilis*; *gangrène scorbutique aux gencives*; *wasserkrebs der kinder*; *water-canker*; *gangrenous aphthæ*. In this form of mortification, the mucous

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membrane of one of the cheeks presents, in some cases, a small superficial ulcer, without pain or discoloration. Sometimes there may be two or three ulcers. In other cases, instead of an ulcer, a small whitish or yellowish-grey spot appears on the mucous membrane, which sloughs, and gives rise to an ulcer similar to the former, or presenting the same colour as the slough by which it was preceded. Nearly at the same time, a greater or less degree of tumefaction of the cheek, opposite the affected part of the mucous membrane, takes place, which increases with great rapidity, and soon extends to the eyelids and lips. The skin of the swollen parts is pale and glistening, resembling wax, hard towards the centre of the swelling, and elastic. By-and-bye this part presents a dull yellowish-grey colour, then becomes black, and sloughs; the whole substance of the cheek undergoes the same successive changes, and in the course of a few days, the cheek, lips, and eyelids are converted into a soft putrid mass, which, falling off, destroys sometimes nearly the whole of one side of the face, lays open the cavity of the mouth, and exposes the gums in a state of sphacelus, the superior and inferior maxillary bones denuded or necrosed, and deprived of their teeth.

From these the physical characters of this form of mortification, together with the state of general debility, and the absence of inflammation, there can be no doubt as to the propriety of including it in the present division of our subject.

MORTIFICATION FROM THE VIOLENT OPERATION OF MECHANICAL, CHEMICAL, AND PHYSICAL AGENTS.

The mechanical agents which occasion mortification are violent blows and contusions; the chemical, powerfully stimulating substances; and the physical, extreme heat and cold. All these agents produce the same ultimate effect in the part of the body which has been submitted to their influence; that is to say, they deprive, to a greater or less extent, such a part of those properties on which its existence depends. Their destructive effects are not, however, always the same in degree and extent, nor are they always produced in the same manner. In one case there may be no gradation of change, no intermediate state of disease which separates these destructive effects from the previous healthy condition of the tissues in which they are produced: local death may be the immediate consequence of the violent operation of such agents. In another, the vitality of the part may not be entirely destroyed. In this case, recovery may or may not take place, the result being dependent on the degree of congestion and inflammation by which the local injury is succeeded.

I. *Mortification from the violent Operation of Mechanical Agents.*—With regard to the physical characters of this form of mortification, and in so far as they depend on the immediate effects of the mechanical cause, it is hardly necessary to observe that they can have no essential or definite form. Laceration, congestion, effusion of blood may or may not be present in the part which has received the injury, the extent and degree of which can seldom be estimated till after the occurrence of inflammation. When this supervenes, which is always the case, it presents appearances similar to those by which it is accompanied when it occurs idiopathically and proceeds to gangrene and sphacelus.

II. *Mortification from Intense Heat.*—The effects of intense heat on the surface of the body are a greater or less degree of excitement, a temporary or permanent suspension of the functions of the part to which it has been applied; or in other words, there is produced a state of inflammation, gangrene, and sphacelus. The essential phenomena of the inflammation and gangrene are the same as those which have already been described, if we except the rapidity of their progress, and also the more sudden and

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extensive developement of phlyctenæ and bullæ, or blisters, particularly when the heat is applied by means of a fluid. The state of sphacelus presents several very characteristic appearances. The skin is of a yellow, grey, brown, or black colour; dry and hard; sunk below the level of the surrounding surface, and quite insensible. These are, sometimes, the only appearances which are at first perceived to follow the action of intense heat, and are certain indices of the complete death of the skin to a greater or less depth. The deeper-seated tissues may also be deprived of their vitality, but to what extent cannot be determined by any change which the cutis may have suffered. The inflammatory redness which succeeds to this state appears almost immediately, and indicates by the rapidity of its course and the peculiar colour which it assumes, the extent both of the gangrene and sphacelus, which could not be determined by any previous change of the cutis produced directly by the heat. The extent of the sphacelus may be said to be always increased by the subsequent inflammation; and parts that were only in a state of gangrene are, by means of it, converted into a state of sphacelus. Whether the state of sphacelus may have been produced by the action of the heat or the subsequent inflammation, the limits of the disease are seldom defined before the end of eight or ten days. The dead are then separated from the living parts, and an abundant suppuration takes place from the denuded surface. The solution of continuity is often imperfectly repaired in consequence of the exuberant production of granulations, which, instead of acquiring the organization of the cutaneous texture, assume that of contractile tissue, which often gives rise to great deformity of the parts with which it is connected.

III. *Mortification from Cold.*—The local and general effects of severe cold are, in many respects, very similar to those produced by intense heat. If the degree of cold be not very great, the circulation and temperature of the skin are increased, as is shewn by this tissue assuming a redder colour, and feeling warmer than before. On the contrary, if the cold be very intense, it may not give rise to any appreciable degree of local excitement; the vitality of the skin and even of the deeper-seated tissues at the same time, may be either greatly reduced or entirely destroyed by the direct operation of this physical agent. There is, however, this difference between the local effects of heat and cold, viz. that the former may produce complete disorganization of the tissues submitted to its action; whereas the latter never produces such a change. Under the operation of the former the local redness rapidly increases, under that of the latter it rapidly diminishes; and in the same manner does the sensibility increase and decrease under the influence of these agents respectively.

By far the most frequent occurrence of mortification is when a frozen limb is exposed to natural or artificial heat, as before a fire or during thaw. The blood that was before frozen thus regains its fluidity, and gangrene and sphacelus, if they were not previously, are now more or less rapidly induced. Under these circumstances, the skin assumes a dark red or livid colour, when it is in a state of gangrene or sphacelus, and the neighbouring skin acquires an erythematous blush, accompanied with a prickling or tingling sensation and a feeling of weight or stiffness in the limb. By-and-bye phlyctenæ appear on the inflamed part of the limb, and grey, livid, or black spots, when it is in a state of sphacelus. Then, also, putrefaction commences and extends till its progress is arrested by the adhesive inflammation.

IV. *Mortification from Stimuli.*—The stimuli included under the present head, and which give rise to mortification, are those which exercise a chemical influence on the tissues with which they come in contact. The nitric, muriatic, and sulphuric acids may

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be cited as examples of chemical stimuli which produce local death when they are applied to the skin or mucous membranes of the digestive organs. The local effects to which they give rise are very similar to those occasioned by intense heat. Like the latter physical agent they instantly destroy, to a greater or less depth, when strong, the cutaneous and mucous tissues. But instead of complete local death or sphacelus, we may have as the result of their action, when diluted, a state of gangrene, or simply inflammation, and which may, as in similar states produced by other causes, terminate in sphacelus.

The local effects of these chemical stimuli are not often confined to one portion of the cutaneous or mucous surfaces; they are generally perceived on several portions of both, the form and extent of which are subject to great variety, more especially in the stomach; the states of vacuity and fulness, or the nature of the contents of this organ, modifying considerably both of these local circumstances. The changes of colour are either yellow, yellowish brown, brown, or black, and seem to depend much on the quantity of blood contained in the part, and the strength of the acid which has been applied to it. In the skin, various degrees of inflammatory redness may be observed to form around the spots, and vesicles are occasionally produced by the effusion of serosity under the cuticle. In the digestive organs, more especially in the stomach, inflammatory redness, extreme congestion, and even sometimes effusion of blood, accompanies the discolouration and disorganization of the mucous and other tissues of this organ, and form an areola of various extent around the latter, the charred appearance of which contrasts strongly with the bright red colour of the former. The inflammatory redness and congestion surrounding the charred parts, or a solution of continuity, from sloughing having taken place; an increase in the thickness and often in the consistence of the edges of the latter; the occurrence of the original lesion in a circumscribed form, and as it were indifferently, in any portion of the stomach, are circumstances which, independently of similar effects of the acid being present in the gullet, mouth, or skin, enable us to distinguish between the morbid appearances produced in the stomach by acid poisons, and the discoloration, softening, and perforation which take place after death in this organ, from the chemical action of the gastric juice.

MORTIFICATION FROM THE DELETERIOUS INFLUENCE OF CERTAIN POISONS.

The poisonous substances which give rise to mortification are either natural or morbid products, derived from the animal and vegetable kingdoms. The former consist in a peculiar healthy secretion of certain animals, vulgarly termed *venom*; the latter are generated by a state of disease of the animal solids and fluids, and are called *virus*. The deleterious agent formed by the decomposition of animal matter, and by that diseased state of rye which gives rise to mortification, has received no specific appellation.

I. *Mortification from a deleterious Agent generated in healthy Animals.*—This form of mortification is often observed to follow the bite of the *cobra di copella*, the rattlesnake, and the viper. When the poison of these animals is inserted into the cutaneous and cellular tissues of one of the limbs, the most acute pain is produced, followed by œdema, swelling, and hardness. If there be any redness of the skin around the wound, it is of short duration, and is succeeded by a livid discoloration, which increases in extent, followed by the formation of phlyctenæ, and diminution of temperature of the part. The hard œdematous swelling of the skin and cellular tissue then becomes soft, crepitates when pressed, and a sanious discharge of a fœtid odour runs out from the

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wound. Such are the local effects of the poison when it possesses a degree of virulence sufficient to destroy the vitality of the part. In milder cases they are limited to a certain degree of œdematous swelling, accompanied by inflammatory redness of the skin, which, being generally greater than in the former case, shows that the occurrence of sphacelus is essentially determined by the primary effects of the poison on the vital properties of the tissues to which it is applied.

II. *Mortification from a deleterious Agent generated during the Decomposition of animal Substances, and in Animals in a State of Disease.*—The production of mortification from a poison derived from these sources is by no means a rare occurrence. It is observed in various internal organs of the bodies of those who have died from mortification of some external part, as the foot or leg, from whatever cause arising, whether from inflammation, a mechanical injury, or a surgical operation. It appears to depend on the septic agent, generated in the sphacelated tissues, being taken into the blood, by means of which it exercises its destructive influence on particular organs, frequently giving rise to gangrene or sphacelus without any perceptible intermediate change. The tissues of the affected organ may not exhibit any of those alterations which constitute the anatomical characters of inflammation. They present, on the contrary, appearances similar to those observed on the external surface of the body to which a virulent poison has been applied, viz. a circumscribed dull deep red, livid, brown, or black colour, in which state they feel firmer than natural; or they are of a dirty grey colour, of a soft pulpy consistence, or entirely decomposed and transformed into a grey or reddish fluid of the consistence of pus, around which the other tissues may not present any material alteration. These appearances are most frequently observed in the lungs and liver. They may exist in several points of these organs at the same time; may occupy a considerable extent of surface varying from the fourth of an inch to two or three inches; and are much more frequently situated towards the circumference than in the central parts of these organs. Their most frequent seat in the lungs is beneath the pleura, and if the patient survives a certain length of time the contaminating influence of the poison to which they owe their origin, this membrane sloughs and pneumothorax is produced. These morbid states of the lungs are not unfrequently accompanied by the deposition of pus, the mode of formation of which will be explained in the fasciculus on *Suppuration*.

The production of what is called *hospital gangrene* affords another example of a septic agent being generated in a morbid condition of the solids, and giving rise to a similar disease when communicated from one individual to another, by means of the dressings or other direct modes of transmission.

Another form of mortification produced by a septic agent generated by disease is that which is called *pustule maligne*, or *charbon* by the French. It is generally believed to originate in horned cattle, among which it sometimes prevails epidemically to a great extent, and that when it occurs in man it is always derived from such animals. The parts of the body on which it is generally observed are those which are usually uncovered, as the face, neck, breast, and shoulders; the hands and arms, feet and legs, parts in fact which come in contact with the skin, blood, or flesh of the affected animals. There are numerous facts which prove that malignant pustule may be thus communicated, and that the blood is strongly impregnated with the septic principle generated in this disease, for parts of the body on which this fluid has been deposited have soon after been affected with malignant pustule, and similar effects follow its injection into the veins.

Malignant pustule commences in the form of a small vesicle, filled with a somewhat bloody serosity, accompanied with a circumscribed œdematous swelling of the skin and

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cellular tissue, which soon extends in breadth, followed by an erysipelatous redness of the skin. As the swelling increases, the skin acquires a glossy aspect, and presents here and there small and large phlyctenæ. The redness soon assumes a livid tint; the central portion becomes brown or black, hard and insensible, whilst the surrounding parts are tense and emphysematous. These changes are produced with more or less rapidity, and sometimes spread to a considerable extent, followed by extensive sloughing of the skin and cellular tissue.

The *carbuncle of plague* presents precisely the same local characters as those of malignant pustule, and seems to owe its origin to a similar cause, viz. the development of a septic agent during the progress of this disease.

It would appear that a septic agent is generated in *various grains in a state of disease*, but more particularly in *rye*, which, when used as an article of food, has frequently given rise to one of the worst forms of mortification. The symptoms which accompany mortification from this cause present considerable variety. In one series of cases, mortification is accompanied or preceded by vertigo, drowsiness, and a malignant form of fever; by a sensation of numbness in the legs, which become afterwards painful, slightly swollen, but not inflamed. The skin is cold and livid, and the sphacelus commences in the centre of the limb, and does not reach the skin till some time after. In a second series of cases, the sphacelated parts are dry, livid, or black; these appearances commencing in the toes and extending gradually upwards, sometimes as far as the thighs. In a third series of cases, the disease commences with lassitude and a sensation as of insects creeping under the skin, and without any febrile symptoms. Soon afterwards the extremities become cold, pale, wrinkled, and benumbed, and at last quite insensible, and incapable of motion; afterwards acute pain is felt, referable to the central parts of the limb. There is now fever and headach; pain extending from the hands and feet to the shoulders, legs, and thighs; and lastly the affected parts become dry, shrunk, and black, and drop off at the joints. Entire extremities are thus separated from the body without hemorrhage. Lastly, in other cases, the chief symptoms are, at first, spasmodic contractions of the limbs, afterwards great weakness of mind, voracity of appetite, which generally terminate in fatuity, and sphacelus of some of the limbs. The parts most frequently affected with gangrenous ergotism, as it is called, are the inferior extremities.

From the above description of the local and general effects of spurred rye, it is by no means easy to say in what manner this poisonous article of food operates so as to produce mortification. One thing, however, is certain, that it is not by an inflammatory process of the parts which become the seat of the disease. That cessation of the circulation, loss of the sensibility and motion of the limb observed to take place at an early period of the disease, are not the consequences of inflammation, is clearly proved by the characters of the local if not the general symptoms which have been detailed. All the local changes appear to be produced as direct consequences of the spurred rye, acting through the medium of the blood or nervous system, or both at the same time; for we have seen that the dead parts are separated without hemorrhage, and it is stated that the blood when taken from a vein is dark and so very thick that it only oozes out from the orifice of the wound.

In several animals that died after having been fed for some time on spurred rye, and that had presented several of the symptoms already mentioned, gangrenous spots were, it is said, found in the stomach, intestines, and liver. But the morbid anatomy of this disease is extremely imperfect, and does not enable us to offer any satisfactory explanation, either in regard to the nature of the primary local lesion or its complications.

DESCRIPTION OF THE PLATES.

PLATE I.

This plate represents perforation of the intestines and pleura, as the consequence of mortification from acute and chronic inflammation. *Fig. 1* represents a portion of the ileum laid open, the glandulæ solitariae and agminatae of which were the seat of acute inflammation, ulcerated or in a state of sphacelus. Several of the solitary glands, AAAA, are seen in the figure surrounded by an inflamed, elevated border, enclosing a yellowish grey slough, formed by the submucous in some, by this and the muscular tissues in others. Several of the aggregated glands were in a similar state, and one of them had entirely sloughed, together with the muscular and peritoneal coats to the same extent, thus giving rise to a perforation, B, of the intestine an inch and a half in length and half an inch in breadth. C, the sharp edges of the perforation; DD, the solitary follicles enlarged. *Fig. 2* represents perforation of the ileum from sloughing of one of the agminated glands, the consequence of an acute attack of inflammation supervening on chronic disease of these glands. A, a portion of the sphacelated coats of the intestine still adhering to the edges of the perforation, the elevated border of which, B, is of a rose-red colour; C and D represent the chronic state of disease of the agminated glands. *Fig. 3*, perforation of almost an entire agminated gland, succeeding also to a chronic state of disease. The edges of the perforation, A, are as smooth as if they had been cut, and present no traces of inflammation. This is the form of perforation which succeeds to chronic ulceration of the follicles, followed by sloughing of the serous membrane, in the manner already described. B, a small portion of the diseased gland remaining; C, another gland in a state of chronic inflammation and ulceration. *Fig. 4*, sloughing and perforation, from chronic tubercular ulceration of the same glands. A, the elevated border of the ulcer infiltrated with tuberculous matter; BB, the submucous and muscular coats in a similar state; C, the peritoneum in a state of sphacelus; D, the tuberculated border of another chronic ulcer; E, the muscular coat, and F, the peritoneum laid bare, the latter of which is perforated at G. *Fig. 5* represents the external appearance of sloughing of the peritoneum, from chronic tubercular ulceration. A, a probe passed through a small perforation occupying the centre of the slough B. *Fig. 6*, sloughing and perforation of the pleura in tubercular phthisis. A, a portion of the pleura in a state of sphacelus, but not yet separated; B, perforation effected and a communication established between the cavity of the pleura and bronchi; CCC, coagulable lymph from the subsequent pleuritis; D, tuberculous matter in the lungs.

PLATE II.

Fig. 1 and *2* represent the appearances of gangrena senilis. In the latter is shown the discoloration of the toes at the commencement of the disease; and in the former,

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the same state in its progress towards the foot. AA, (*Fig. 2*.) three of the toes nearly black and shrunk; B and C, the small and great toes partly of the same colour and presenting a slight tinge of red. A, (*Fig. 1*.) the same black discoloration gaining the upper surface of the toes, the skin of which, as well as that on the back of the foot, B, is wrinkled; C, congestion of the veins. *Fig. 3*, another example of gangrena senilis commencing in the toes, A, a little beyond which the mortification was arrested at B, by the adhesive inflammation, and a partial separation effected at C. *Fig. 4*, mortification of the skin and subjacent cellular tissue from an obstacle to the return of the venous blood in consequence of disease of the heart. In this figure the mottled capillary congestion of the skin is well marked. A, the epidermis raised by the effused serosity; B, the sphacelated cutis; D, the cutis and cellular tissue in the form of a slough; D, the same parts in a similar state, which are partly separated at E, showing the great depth of the solution of continuity which follows, owing to the serous infiltration of the subcutaneous cellular tissue.

PLATE III.

Fig. 1 and *2* represent the state of the bloodvessels in two of the forms of mortification delineated in the preceding plate. *Fig. 1* represents the femoral vein A, and artery B, laid open, of the case *Fig. 4*, Plate II. The femoral vein, from the inferior third of the thigh, was filled with a dark-coloured firm coagulum C D, extending downward through all its branches as far as they could be traced. All the other veins of the leg were similarly obstructed. The inferior third of the artery was also obliterated by a red fibrinous coagulum, E, which terminated abruptly above at F, and below at G, where it divides into the anterior and posterior tibials. At these two points the colouring matter had been absorbed, and adhesion, H, was taking place between the fibrinous matter and the lining membrane of the artery. *Fig. 2* shows the morbid condition of the arteries in the case of gangrena senilis represented in *Fig. 1* and *2*, Plate II. A, inferior portion of the abdominal aorta laid open; BB, common iliacs; C, internal, D, external iliacs; E, femoral, F, popliteal, G and H, anterior and posterior tibial arteries. These two latter arteries, as well as their branches, were converted into solid cords at KK, from the presence of a fibrous and fibro-cartilaginous tissue developed either in the walls or in fibrine contained within them. The capacity of the popliteal and femoral arteries was considerably diminished by irregular portions of fibrine, projecting from their internal surface, which presented throughout the greater part of its extent cartilaginous and ossific deposits, forming plates, spicula, and fissures of various dimensions. Within the aorta, the fibrine of the blood had accumulated around some of these spicula in the form of flat pyriform tumours. They were attached by a narrow neck, presented a radiated conformation, and floated in the cavity of the vessel in the direction of the circulation of the blood, the course of which they must have impeded to some extent. *Fig. 3* is a beautiful example of mortification of the intestine from intus-susception, in the manner already described. A, superior portion of intestine; B, the inferior portion entire; C, a part of the same laid open, to show the invaginated intestine D, in a state of extreme congestion, and thrown into folds at E. The mechanism of the congestion, gangrene, and sphacelus is pointed out by the condition of the vein F, which is seen to be drawn in along with the invaginated intestine, and compressed between the latter and the upper border of the inferior or invaginating portion of

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intestine. *Fig. 4* represents that form of mortification sometimes observed in the lungs, occasioned by obliteration of the arteries and veins, in consequence of chronic induration of the pulmonary tissue. AA, a cavity of considerable size, formed by sloughing of the indurated pulmonary tissue; B, a section of this tissue, which was as firm as fibrous tissue, composed of grey-coloured substance, strongly impregnated in some parts with black pulmonary matter; C, a portion of the sphacelated pulmonary tissue, attached to obliterated branches D, of the pulmonary artery F, containing at EE firm coagula. G, obliterated veins and bronchi; H, a bronchus communicating with the excavation. *Fig. 5* represents mortification of the lungs succeeding to gangrene of the lip, which was said to have been occasioned by the bite of an insect. AA, the surface of the pleura covered with flakes of coagulable lymph; B, congestion of the pulmonary tissue beneath the pleura, and confined to a single lobule, the surrounding cellular tissue being in a state of sphacelus; C, another lobule and surrounding cellular tissue in the state of sphacelus; B, puriform matter situated beneath the pleura, which at E is accompanied with great congestion. F, a section of the lung presenting similar appearances; GG, great congestion of individual lobules with sphacelus of their surrounding cellular tissue; H, a lobule converted into a soft, straw-coloured, puriform substance, which, in another lobule at K, has been removed, leaving a small cavity in its situation, having the appearance of an abscess.

PLATE IV.

Fig. 1 represents mortification of nearly the inferior third of the upper lobe of the left lung, limited by adhesive inflammation. C, a large cavity, lined by a yellowish grey-coloured thick membrane DDD, united with the pleura of the upper surface of the inferior lobe A, and with the pulmonary tissue of the upper lobe B, which was in a state of grey hepatization F, to the extent of about a quarter of an inch in depth. Beyond this the pulmonary tissue G was quite healthy. The sphacelated substance of the lung EE, thus isolated and enclosed in a membrane of new formation, was of a dirty yellowish brown or black colour, of a pulpy consistence, composed of shreds of cellular tissue and obliterated bloodvessels, and a considerable quantity of an offensive grumous fluid, similar to that which had been expectorated for some time before death. Several of the bronchi communicated freely with the cavity, in which *gargouillement* was very strongly marked. *Fig. 2*, a portion of skin, from a weak scrofulous child, to which leeches had been applied. The edges of the leech-bites, A B, of a purple colour; C, the dark inky appearance which they afterwards present, succeeded by circumscribed sloughing of the cutis. *Fig. 3*, mortification of the inferior lip, A, from the bite of an insect. The appearances represented in the figure, however, were produced by cauterization, which was followed by inflammation, the commencement of which is seen around the eschar and proceeding towards the neck, B. *Fig. 5*, Plate III. represents the state of the lung in the same case.

Platel.

Fig. 2.

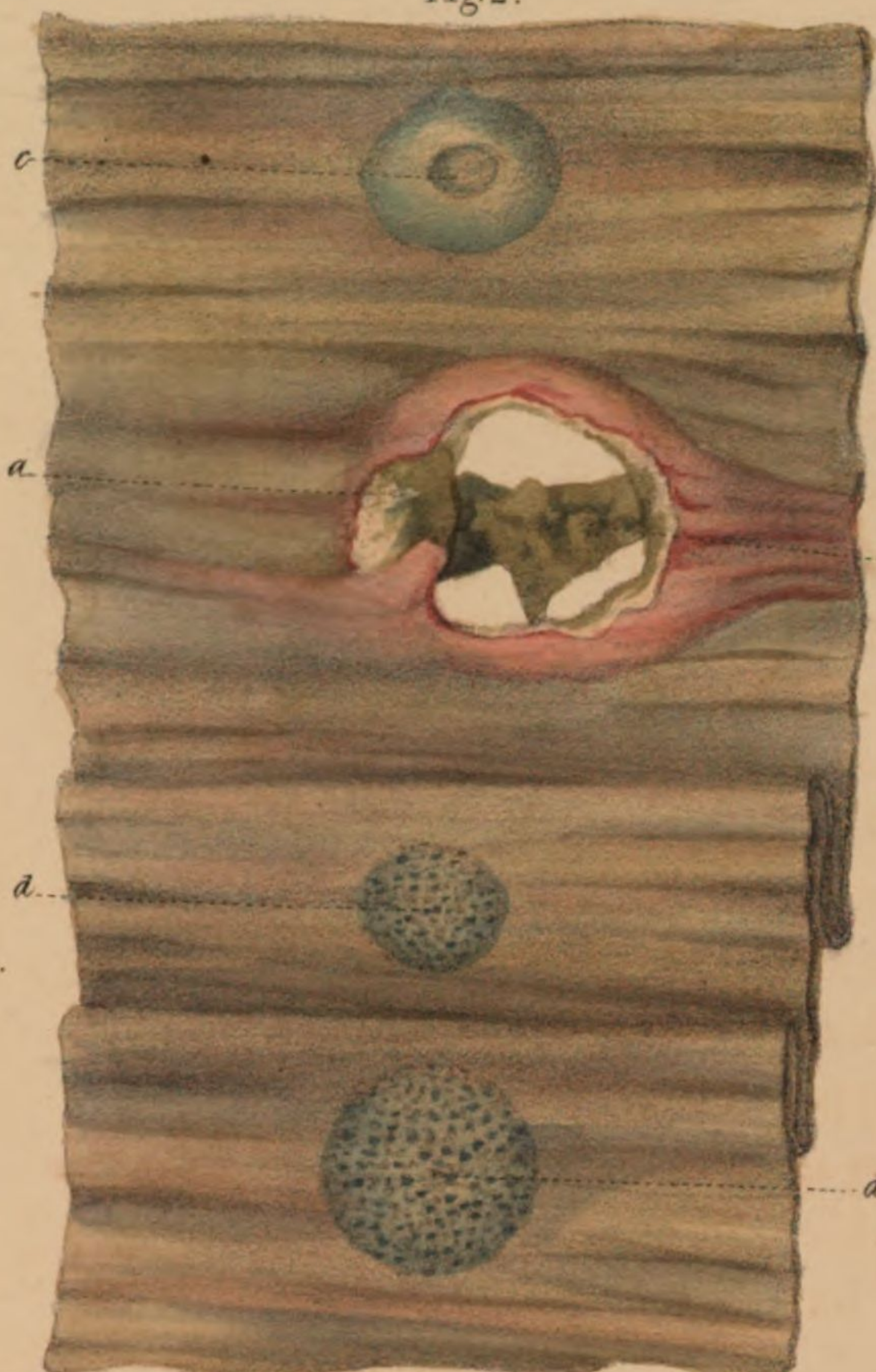


Fig. 1.

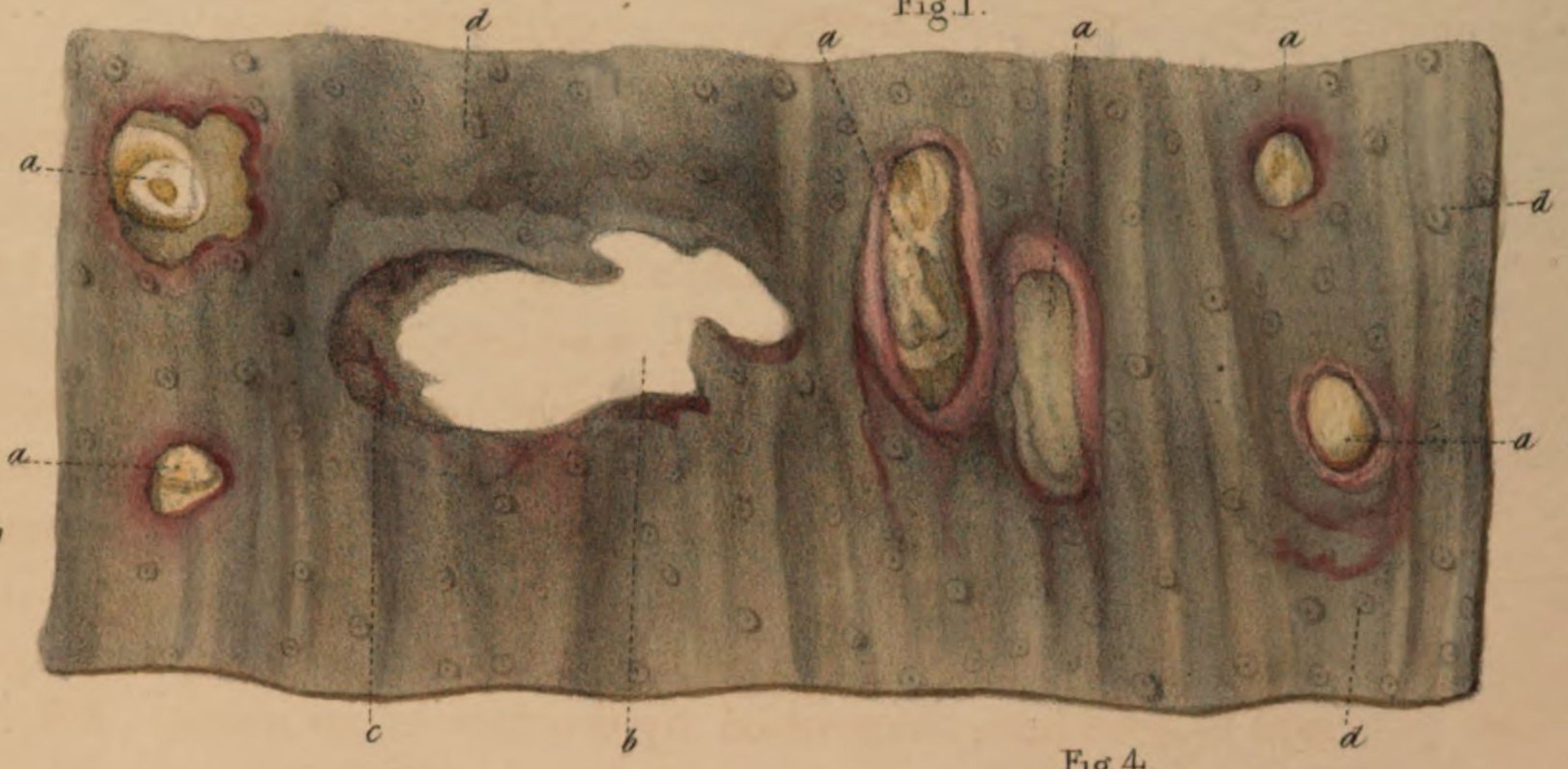


Fig. 4.



Fig. 5.



Fig. 3.



Fig. 6.



Plate II.

Fig. 3.



Fig. 4.

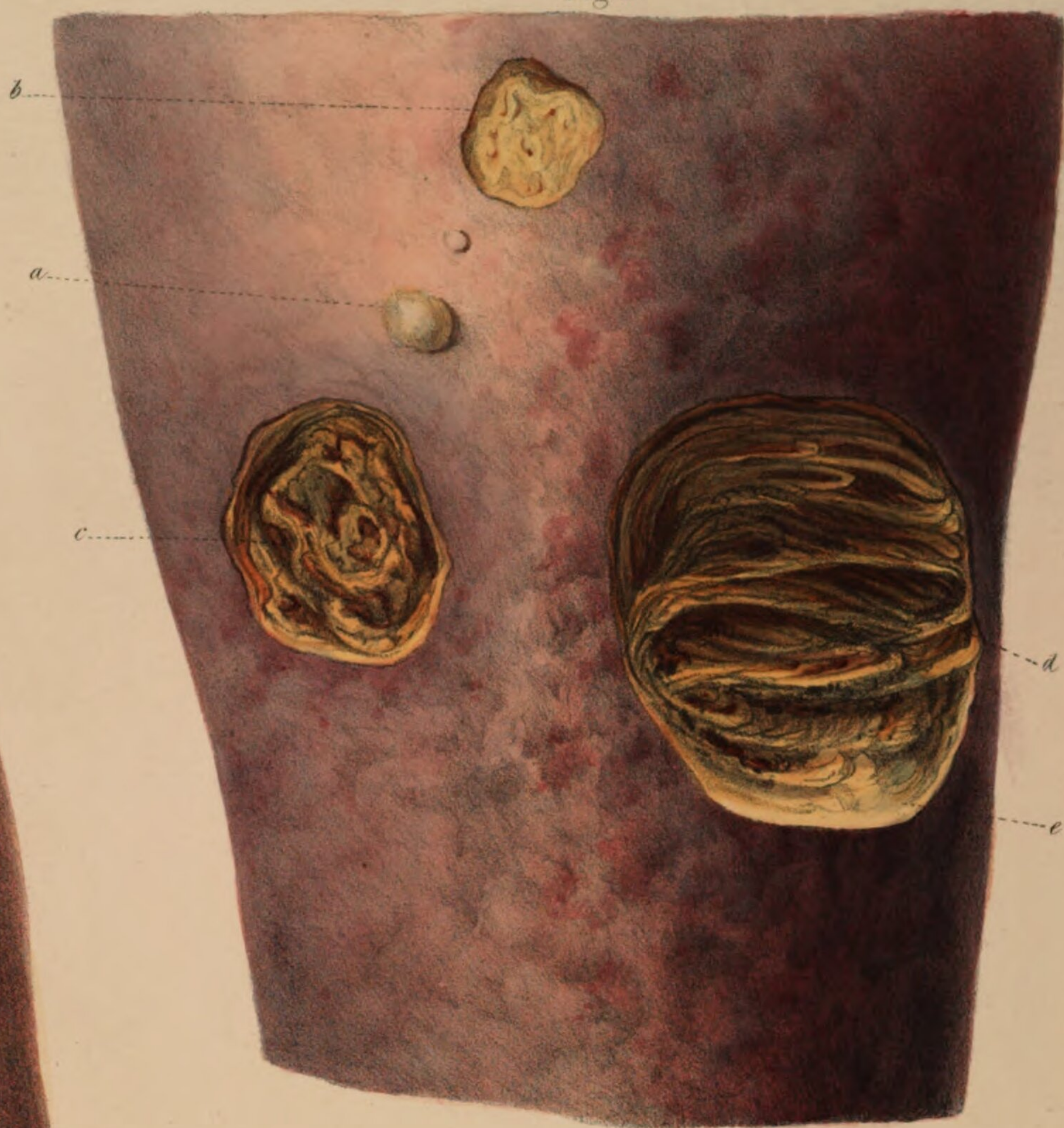


Fig. 2.

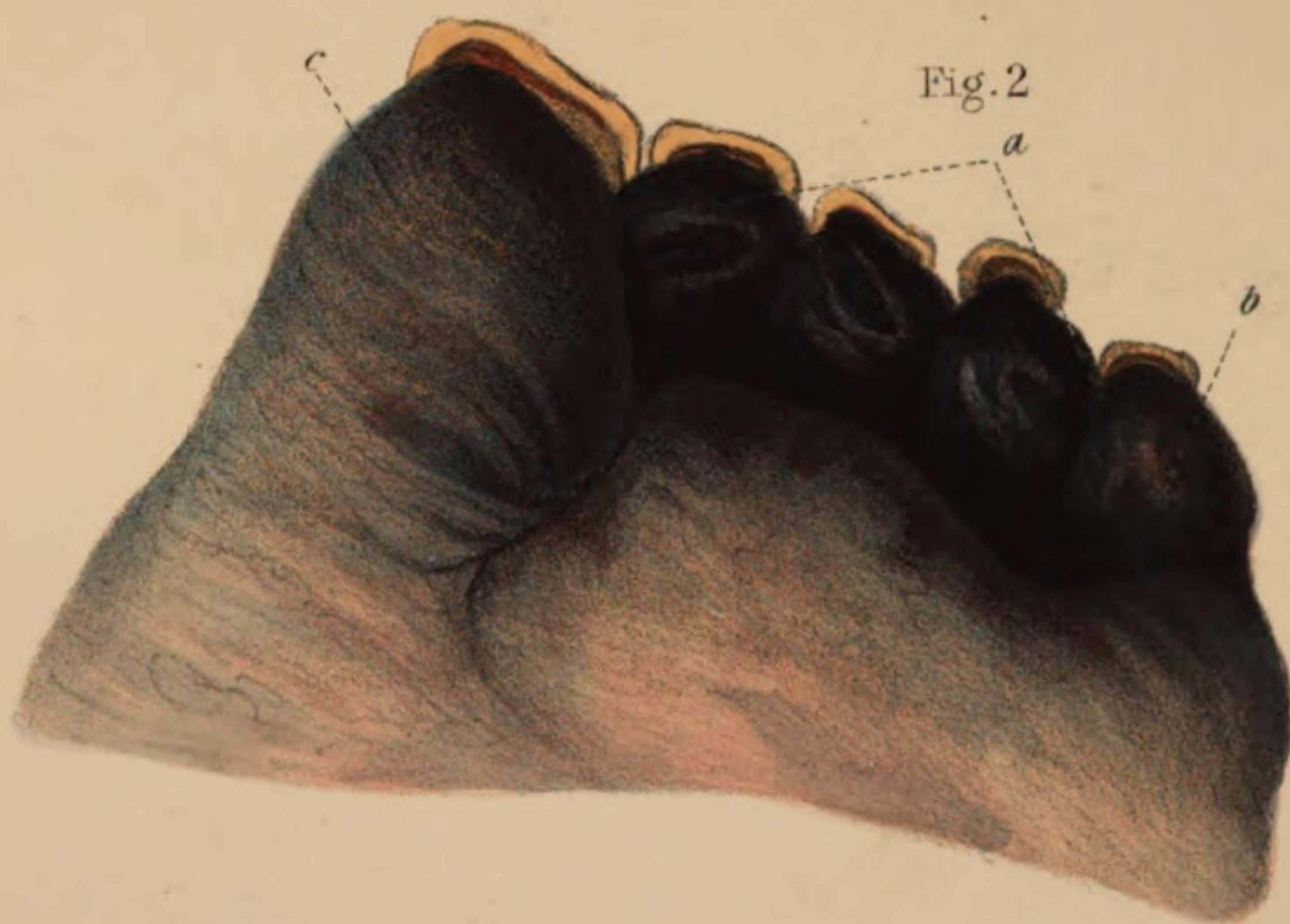


Fig. 1.



Plate III.



Plate IV.

Fig 3.



Fig 2.



Fig 1.



HEMORRHAGE.

HEMORRHAGE, in the more accurate and now generally received acceptation of the term, consists in the extravasation of blood, or the escape of this fluid during life from the vessels in which it is contained and circulated, into the substance or on the surface of organs, whether it be retained in these situations or conveyed to the external surface of the body.

Hemorrhage may take place from the heart, arteries, veins, and capillaries, as the immediate consequence of a solution of continuity occasioned by incised wounds, puncture, laceration, ulceration, and mortification; or the sanguineous discharge may proceed from the capillaries, which present either no perceptible lesion of structure, or merely an increase of capacity, whereby the red globules of the blood are permitted to pass along these vessels with the other constituents of this fluid which is poured out after the manner of exhalation. Hemorrhage may also take place from anormal vascular tissues, developed in various parts of the body, and deriving their supply of blood from the organs in which they are situated. Of this kind are the cephalomatous forms of carcinoma, and erectile tissue. In the former, bloodvessels exist in great abundance, which are often laid open by ulceration and mortification. In the latter, which are composed of a congeries of arteries and veins, either of which may predominate, or which present a structure resembling that of the spleen, the occurrence of hemorrhage is likewise sometimes observed from similar causes.

Besides these the immediate causes of hemorrhage, there are others which exercise a powerful influence in the production of this lesion, and without the existence of which it would often not occur. Such are, more especially, various morbid conditions of the heart and vascular system. Those of the heart consist principally in hypertrophy, or hypertrophy and dilatation of its walls, and in the non-occlusion of the auriculo-ventricular orifices, especially on the left side of this organ, in consequence of permanent retraction of their valves. Under the mechanical influence of these morbid states of the walls and valves of the heart, one uniform result follows, viz. venous congestion, the necessary consequence of an obstacle to the return of the venous blood. The congestion thus produced becomes, frequently from unknown causes, much greater in some organs than in others. It increases slowly or with great rapidity, either within a circumscribed space, as in the brain and lungs, or over a large extent of surface, as in the digestive mucous membrane. At length the capillaries, incapable of further distention, are ruptured, and the blood is effused into the surrounding cellular tissue; or these vessels, in consequence of the dilatation which they have undergone, afford an exit to the blood in the form of exhalation from membranous surfaces. The diseased conditions of the vascular system which favour the production of hemorrhage consist in the deposition of calcareous and ossific matter in the coats of the arteries; in softening of these coats; and in the mere loss of elasticity of their lining membrane. The dis-

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eased coats of the arteries, no longer capable of counteracting the force with which they are distended by the blood, either undergo the successive changes which characterise the progress of aneurism, or are immediately ruptured.

There are several circumstances connected with these morbid states of the coats of the arteries which deserve notice, as they sometimes serve as a means of enabling us to ascertain with greater facility and certainty the immediate cause of hemorrhage in certain organs: such are the much more frequent occurrence of these lesions in the large than in the minute divisions of the arteries; their frequent occurrence in some organs, as the brain; their entire absence in others, as the lungs and mucous membranes; and the period of life at which they are most commonly met with. Hemorrhage, in consequence of the immediate rupture, and of the previous formation of an aneurism from disease of the coats of an artery, presents, likewise, a striking difference in the relative frequency of its occurrence, as produced by these two lesions. In the brain, few cases of hemorrhage have occurred from aneurism, whereas, in the same organ, the examples are numerous in which it has been traced to rupture. The reverse of this happens in other organs, or rather in every other part of the body the arteries of which are subject to the same diseases as those of the brain,—a difference which seems to be satisfactorily explained by the following circumstance, viz. the almost entire absence of a cellular sheath in the arteries of the former, and its greater or less abundance in those of the latter.

I have already alluded to the state of local congestion which precedes hemorrhage, in consequence of an obstacle to the return of the venous blood, in various diseases of the heart. But there are other forms of local congestion which precede hemorrhage, of a very different nature from the former, such as those which take place in the most depending parts of the body in persons advanced in age or debilitated by disease; and in the substance of various organs, without the obvious concurrence of these causes, and without any other lesion than extreme fluidity of the blood, or the almost total absence of that plastic property by means of which the coagulation of this fluid is effected in the healthy state. In the former the hemorrhage is seldom considerable, and is chiefly confined to the subcutaneous or intermuscular cellular tissue; in the latter it is often extensive, and affects several organs at the same time or in succession, as in some cases of scorbutus and purpura hemorrhagica.

The last form of hemorrhagic congestion to which I shall allude, is that which takes place vicariously of a similar natural or morbid state in a remote organ. In vicarious or supplemental hemorrhage, we have the most convincing illustration of sanguineous exhalation, or of hemorrhage occurring without rupture; whilst at the same time there hardly can remain a doubt, that the capillaries are the sole agents concerned in the actual production both of the increased flux of blood which constitutes the state of congestion, and of the hemorrhage which follows. Nor does there appear any reason for supposing that the modification of function which the capillaries undergo in vicarious hemorrhage, differs in any respect from that which takes place in these vessels before and during the periodical sanguineous discharge from the uterus. Anormal erectile tissue furnishes striking examples of congestion and hemorrhage, produced under the immediate influence of a functional operation of the vessels of which this tissue is composed.

From these general observations on the several forms of hemorrhage, it would appear natural to refer the immediate occurrence of this disease to the existence of two essentially distinct lesions, the *physical* nature of one of which is ascertained by direct observation; and the *vital* nature of the other not less satisfactorily determined by

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observation and analogy. On these two distinctive characters of hemorrhage, the following classification of its several forms may be established.

HEMORRHAGE FROM PHYSICAL LESIONS.

I. *From Solutions of Continuity.*

1. Incised wounds.
2. Puncture.
3. Laceration.
4. Ulceration.
5. Mortification.

II. *From a Mechanical Obstacle to the Circulation.*

1. Situated in the heart.
2. Situated in the bloodvessels.

HEMORRHAGE FROM VITAL LESIONS.

I. *From a Modification of Function of the Capillaries.*

1. In vicarious hemorrhage.
2. In hemorrhage from erectile tissue.

II. *From a diseased State of the Blood.*

1. In scorbutus.
2. In some forms of purpura.
3. In some forms of typhoid fever.

III. *From Debility.*

1. In the depending parts of the body.

Before proceeding to describe the physical characters of hemorrhage in those organs in which it most frequently occurs, I shall give a general view of the locality of this lesion, the seat of the effused blood, its quantity, its local effects, and some of the more important changes which are observed to take place in this fluid.

The *locality* of hemorrhage in general is extremely varied, several of its forms, as we have already seen, occurring only in particular organs, whilst others have no restricted locality, although they may take place more frequently in some organs than in others. Age determines a remarkable difference in this respect; hemorrhage from the mucous membrane of the nasal cavities being most frequent in childhood; from that of the air-cells and bronchi, in youth; of the digestive, uterine, and urinary organs, in middle age and the decline of life; and in the brain in old age. Hemorrhage much more rarely occurs in the organs of generation in the male than in those of the female. There are also natural peculiarities of structure which exercise a certain influence in determining the locality and frequency of hemorrhage. The delicate structure of the brain, the loose cellulo-vascular organization of the lungs, are circumstances which seem to favour the occurrence of hemorrhage in these organs, as much as the opposite conditions of structure of the liver, render its production in that organ extremely rare and limited in extent. That structure of organs which enables them to undergo great distention operates powerfully in preventing the occurrence of hemorrhage. This is particularly exemplified in the spleen, which is seldom ruptured, however great the quantity of blood accumulated in it may be, unless it has received the shock of an external injury.

Hemorrhage may take place in a great many organs at the same time or in succession, as in scorbutus and purpura hemorrhagica; or it may be confined to one organ, occupying only a small portion, or the greater part of its substance in one continuous mass of blood; or it may occur in a great many distinct portions of various extent, as in cerebral and pulmonary hemorrhage. There is also some peculiarity of function which determines

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the locality and favours the frequency of hemorrhage, as that of mucous membrane, which is more subject to this lesion than any other *elementary* tissue, whether it occurs in consequence of obvious physical causes, or as a vicarious discharge.

The *seat* of hemorrhage has been before pointed out, viz. the heart, arteries, veins, and capillaries; and the vascular system of certain anormal tissues. That of the effused blood presents several important considerations. This fluid may be poured out into the cellular tissue of every part of the body, into all the serous cavities and mucous passages. In the two former situations it is commonly retained, and in the latter situation rejected, in whole or in part only, by one or more of the external orifices of the body. It sometimes, however, happens that the blood, instead of being retained in the cellular tissue, makes its escape into a neighbouring cavity, as into that of the pleura when this membrane is ruptured in pulmonary apoplexy. The effusion of blood into the ventricles of the brain and theca vertebralis has, in the great majority of cases, a similar origin, viz. rupture of the cerebral substance which forms the walls of the ventricles. The seat of the effused blood may likewise be at a considerable distance from the vessels by which it is poured out, as in the case last alluded to, and in many cases of wounds and rupture. The ejection of blood from the stomach and intestines may have its source in the rupture of an aneurism into the œsophagus, and from the lungs in consequence of a similar termination of this disease in the trachea.

It is on a difference of seat that the division of hemorrhage from the lungs into bronchial and pulmonary has been founded, but which would be more accurately expressed by the terms bronchial and vesicular, inasmuch as the hemorrhage proceeds from the mucous membrane, whether the blood be expectorated, constituting hemoptysis, or retained, and producing pulmonary apoplexy.

But in no organ has so much importance been attached to the seat of hemorrhage as in the brain, either as regards its relative frequency in particular portions of this organ, or the influence which it exercises in the production of paralysis in certain organs and parts of the body.

Effusion of blood within the cranium may take place in the brain or cerebellum, in the crura of these, in the pons Varolii, and in the medulla oblongata; in the corpus callosum; in the ventricles; in the surface of the brain beneath the pia mater; in the cavity of the arachnoid; between this membrane and the dura mater, which it covers; and between the dura mater and cranium.

There has not as yet been established any *special* relation between the seat of the effused blood and the paralysis of particular organs. It has been said that paralysis of the *superior* extremities depends on the effusion taking place in the *thalami*, or in the cerebral substance situated on a *level* with, and *posterior*, to them; and that paralysis of the *inferior* extremities depends on the effusion taking place in the *corpora striata*, or in the cerebral substance situated on a *level* with, or *anterior*, to them. This statement, the accuracy or fallacy of which can be put beyond all doubt by any one who has studied this subject practically, is far from agreeing with the result of my observations. The fact is, there are cases of cerebral hemorrhage in which the paralysis is either limited to, or greatest in degree, in the inferior or superior extremities, and in which cases the effusion bears the respective relations to which I have alluded. But there are others, and they are numerous, which shew that paralysis of the extremities has no *necessary* connexion with effusion into these portions of the brain, inasmuch as they furnish us with examples of paralysis either of the inferior or superior extremities separately, or of both at the same time, when the effusion is confined to the cerebral substance of the

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convolutions, or to the *middle* lobe of the brain. Besides, paralysis of the extremities is as complete in hemorrhage of the *cerebellum* as in that of the brain.

It has also been said that the *loss of speech* which not unfrequently accompanies cerebral hemorrhage, depends on the effusion occupying the *anterior lobes* of the brain, a statement which derives still less support from actual observation than the former; for *blood may be effused in the anterior lobes of the brain without giving rise to any modification of speech whatever*.

The best established facts regarding the seat of cerebral hemorrhage, and the relation which exists between it and paralysis, are the following:—

1. That the paralysis occupies the side of the body opposite to that of the brain or cerebellum, in which the effused blood is situated.

2. That the paralysis affects only one side of the body, when the effused blood is confined to one hemisphere of the brain, or one of the lateral lobes of the cerebellum.

3. That the paralysis exists on both sides of the body when the hemorrhage has taken place in both hemispheres of the brain, or both lateral lobes of the cerebellum; into the ventricles; the pons Varolii; the medulla oblongata; and on the surface of the brain.

4. That paralysis of both sides of the body may also take place when the hemorrhage is confined to one hemisphere of the brain or lateral lobe of the cerebellum, but is so extensive as to produce compression of the opposite hemisphere or lobe.

As hemorrhage of one of the lateral lobes of the cerebellum, like that of one of the hemispheres of the brain, gives rise to paralysis of the opposite side of the body, we should, *a priori*, have expected that hemorrhage of the *left* lobe, for example, of this organ, and of the *right* hemisphere of the brain occurring together, would have given rise to general paralysis, or of both sides of the body. Such, however, as has been observed by Andral, is not the case, for the paralysis is found to exist on that side of the body only opposite to the hemisphere of the brain which is the seat of the effusion, the other side remaining unaffected by the effusion in the cerebellum. This is one of the most remarkable circumstances connected with the influence of cerebral hemorrhage in the production of paralysis, and cannot be explained by any knowledge we possess, either of the structure or functions of the cerebral organs. Nor is this the only circumstance connected with paralysis from cerebral hemorrhage which has eluded our researches on this subject, as we have seen in some of the instances to which I have alluded under another point of view, such as the occurrence of paralysis in the inferior and not in the superior extremities, and *vice versa*, loss of speech in one case and not in another, under circumstances of locality apparently similar, or which afford no rational explanation of such differences.

The *quantity* of the effused blood varies from a few drops to several pints. It is, *cæteris paribus*, generally considered to be greatest when the solution of continuity affects the trunk of an artery, and a ready exit is afforded to the diffusion or escape of the blood. But as the fatal effects of the loss of blood are greatly hastened by the degree of rapidity with which this fluid is effused, the least perceptible lesion of the capillaries over an extensive surface, or a small opening into the coronary artery of the stomach, may be followed by hemorrhage as extensive as that which takes place from a large rupture of an aneurism of the aorta. But there is no circumstance which appears to regulate so much the quantity of the blood effused, as the vital importance of the affected organ.

It is rare to meet with hemorrhage to a great extent from veins of large size,

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except in the case of wounds, and in varix of the extremities accompanied by chronic ulceration of the integuments. In the latter case, extensive hemorrhage may be the result of perforation of a vein not larger than a common quill. I had an opportunity of examining the vein in an example of this kind, the morbid condition of which, and of the surrounding cellular tissue, afforded a satisfactory explanation of the fatal extent of the hemorrhage. The walls of the vein were much thicker than those of an artery of the same size, and were so firmly united with indurated cellular tissue, that a considerable degree of pressure was required to approximate their internal surface. The consequence of this condition of the vein was, that its capacity could undergo no diminution during the hemorrhage, the blood continuing to escape as through an inert tube by the opening which had been effected by ulceration. The patient, who was about forty years of age, expired in the space of little more than ten minutes.

The *local effects* of hemorrhage which require notice are, compression, laceration, obstruction of natural passages, inflammation, suppuration, and mortification.

There is only one organ, the brain, in which the *compression* produced by the effused blood gives rise to any remarkable modification of function, viz. paralysis. It is, however, an important fact that blood, even when effused in considerable quantity within the cranium, may not give rise to any marked symptom of apoplexy. I have met with several cases of this kind, in which the quantity of the effused blood amounted to several ounces. The most remarkable of these cases occurred in a stout man, a porter, of middle age, who stumbled in the street from the effects of liquor, and struck his head against the pavement. He got up immediately, and walked home. A few days after, he felt himself less capable of making considerable exertion than formerly; he could not, as before the accident, raise a heavy load, or walk steadily under a heavy burden. This state increased, but not to such a degree as to prevent him from continuing his daily occupation for three weeks, at which period he presented himself at the Hôtel Dieu of Lyons. His general health was good; his intellectual faculties were entire; he walked and expressed himself with perfect freedom; the pulse and temperature of the skin were natural. Under these circumstances the receiving physician refused to admit him. Next day he was brought to the hospital in a state of complete coma, with stertorous breathing and general paralysis, and died during the day. We found that he had gone home and complained to his wife that he had been refused admission to the hospital because he had no fever; and that she, in order to remove this objection, had administered to him nearly a quart of hot wine containing a quantity of pepper. The result of the autopsy was interesting: at least six ounces of blood covered the superior half of the surface of the brain, but was separated from it by the arachnoid, between which and the dura mater it was enclosed. The blood was partly fluid and partly coagulated, red or almost black; and a false membrane, of the thickness of card-paper, lined the internal surface of the dura mater in contact with the blood, presenting in several points of its extent numerous vessels of new formation. The only lesion observed in the brain itself was congestion of its venous system. These appearances afford a most satisfactory explanation of the apparent anomaly observed in this interesting case, viz. the absence of paralysis during the existence of an extensive effusion of blood within the cranium, as well as of the cause of the general paralysis and death which supervened. The state of the blood and the presence of a false membrane mark the duration of the hemorrhage and its progressive nature. The slow and gradual effusion of the blood in this case had allowed the circulation of the brain to accommodate itself to the presence of so much additional fluid, thereby obviating the effects of pressure.

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So long, in fact, as the quantity of the blood circulating in the brain was not increased beyond the limits within which it was compatible with the presence of the blood that was effused, none of the usual effects of compression were observed; but so soon as the stimulating potion which the unfortunate patient swallowed, had, by its effects on the general circulation, and probably also on that of the brain, occasioned an influx of blood towards this organ, compression must then have taken place as certainly as the general paralysis by which it was almost immediately followed. The slow development of chronic abscess within the substance of the brain, and the gradual accumulation of water in its ventricles, without any accompanying increase of the parietes of the cranium, are lesions perfectly similar in their nature to the former as regards the law of pressure in the production of paralysis, and which law may be thus generally expressed and applied to the disturbance of any special function from this cause,—that the disturbance of the functions of an organ from pressure is in the direct ratio of the rapidity with which the compressing cause operates.

Laceration of the substance of the lung is not a rare occurrence in pulmonary hemorrhage, but is not perhaps produced unless when the quantity of the blood effused in a single portion of the lung is considerable. Laceration of the pleura is much less frequent than that of the lung, and does not, so far as I have observed, occur, unless the pulmonary tissue has previously been lacerated, and the hemorrhage has taken place near the pleura.

The delicate organization of the brain would render it probable, did not the evidence of direct observation show, that hemorrhage of this organ is accompanied with rupture of its fibrous structure. I have, certainly, in a few instances, seen a small quantity of effused blood infiltrated between separate bundles of fibres of the cerebral substance, and even layers of this substance of considerable extent, detached in the direction of its fibrous structure. This is a circumstance in the locality of cerebral hemorrhage which deserves to be investigated; for if a quantity of blood sufficient to produce paralysis can be effused into the substance of the brain without rupturing its fibrous structure, an explanation might be afforded of the perfect cure of paralysis in some cases, and of the more rapid and complete disparition of it in others; and likewise of the absence of any of those formations, such as cysts or cellulo-fibrous cicatrices, which usually indicate the previous existence of cerebral hemorrhage.

The *obstruction* of natural passages by the effused blood is an occurrence which leads to no important modification of function, except in pulmonary hemorrhage. Respiration is impeded to an extent proportionate with the quantity of the effused blood; and the stethoscopic signs of the disease are founded chiefly on the presence of this fluid in the vesicular structure of the lungs, and in the bronchi.

Inflammation, suppuration, and mortification are morbid states which are not often observed to follow the presence of effused blood. Inflammation, terminating in softening, is sometimes met with in the cerebral substance surrounding the effused blood, and is considered as the cause of the convulsive twitchings and spasmodic contraction which occasionally take place in the paralysed extremities before death, or some time after the apoplectic attack. Suppuration and mortification, on the contrary, are sometimes met with as terminations of pulmonary hemorrhage; but the latter is, I believe, confined to persons advanced in age, in whom also mortification of this organ from primary inflammation is almost exclusively observed to occur.

Changes which take place in the effused blood.—This part of our subject embraces, in some measure, the consideration of one of the most important facts in pathology, viz. the

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organization of the blood, or, in more strict language, of the plastic element of this fluid, the fibrine, *per se*. I shall, however, confine myself at present to a relation of those changes which are observed to take place in the effused blood, and which, from the circumstance of this fluid becoming organized, constitute in certain organs one of the modes of cure of hemorrhage and of some of its local effects. When blood is effused in an organ, one of two things follows as regards the future condition of this fluid—it is either removed, or remains and becomes organized. Although the relative frequency with which these changes take place in different organs and tissues has not been determined, it is well known that the one occurs more frequently than the other in certain organs. Thus, blood effused into the subcutaneous cellular tissue is almost always soon removed by absorption; that it is speedily rejected from the digestive mucous passages; that it is sometimes three, six, or eight weeks before its removal is effected from the vesicular structure of the lungs; and that if it is ever entirely removed from the brain by absorption, it is perhaps only under those circumstances of locality to which I have alluded, or when it has been effused on the surface of this organ. On the other hand, the retention and organization of the effused blood have never been observed in mucous passages, as the digestive, urinary, and genital; nor does my observation lead me to believe that this fluid ever becomes organized in the vesicular structure of the lungs. But there are examples of the blood becoming organized when retained in the subcutaneous cellular tissue, and in the cavity of the peritoneum; and numerous have been the opportunities afforded of witnessing this process and its subsequent results after cerebral hemorrhage. As this subject, however, will come before us again under a special form, I shall not now bring forward the facts on which the accuracy of these statements rests. It will, besides, be sufficient for our present purpose to describe briefly the changes which take place in effused blood that becomes organized, and in doing so I shall confine myself to a description of those which are observed in this fluid after cerebral hemorrhage.

The changes observed to take place in blood when effused into the substance of the brain, may be grouped with advantage under the two following heads: 1st, Those which are characterized by modifications in the colour and consistence of this fluid; 2d, by the formation of a vascular tissue. The changes of colour consist in the gradual deepening of the red till it amounts to black, and the successive transitions to brown, dull green, orange, pale yellow, or yellowish white. The consistence of the blood is indicated by the degree of coagulation which this fluid has undergone, and does not exceed that of firm fibrine. It is not until the latter changes of colour have taken place, and the fibrine, separated from the other constituents of the blood, has assumed a fibrous or laminated appearance, that bloodvessels are observed to form in it. It is from this stage, therefore, of the changes observed to take place in the blood, that we date the commencement of the formation of a vascular tissue. Now, the subsequent changes which take place in this tissue are of two kinds. It may either retain for a long time its primitive arrangement, that of fibrine, and afterwards become converted into a firm fibrous tissue, which, gradually diminishing in bulk, is at last reduced to a small circumscribed thin portion, which constitutes the cicatrix of the original lesion of the brain: or, the organized fibrinous substance may be converted into a loose cellular tissue, filled with a serous fluid, and is generally traversed by a considerable number of bloodvessels. As the quantity of the serous fluid increases, that of the cellular tissue diminishes, as well as the number and size of the bloodvessels with which it was before provided. In this manner a cavity of considerable extent is formed, filled with serosity of a citrine colour, and bounded by the remaining cellular tissue in contact with the

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substance of the brain. It is this portion of the cellular tissue which appears to be transformed into the serous membrane which afterwards lines the entire surface of the cavity, and which converts it into what is commonly called the *apoplectic serous cyst*. The obliteration of this cyst is the next circumstance which takes place in the progress of cure. This is accomplished by the gradual removal of the serosity contained in the cyst, and the consequent approximation of its walls, which become united and form a cicatrix. Lastly, the cicatrix itself, whether formed in this manner or in the manner previously described, disappears; such, at least, seems to happen in some few cases of paralysis which have undergone a complete cure; and where an opportunity has been afforded of examining the brain, the only morbid appearance observed in this organ consisted of a change in the bulk and direction of a circumscribed portion of the grey and white fibrous structure of one of the thalami or corpora striata.

Such appear to be the two modes which nature employs to accomplish the cure of a solution of continuity occasioned in the brain by an effusion of blood. That the paralysis generally diminishes during the progress of the curative process; that the degree of diminution which it undergoes corresponds sometimes with the more advanced stages of this process, and that it completely disappears with the cicatrization of the original lesion, are facts well established. But the cases in which these relations are observed are far from being numerous, the same stage of the curative process—a cyst, being often found in cases of severe paralysis which had never undergone any sensible diminution; in others in which it had nearly disappeared, and in some few in which no traces of it were observable several years before death. Some of these differences with regard to the degree of the paralysis may depend on a difference in the seat of the original lesion, the extent of this, and the degree of injury done to the fibrous structure of the brain. But how far these or other circumstances will be found sufficient to remove the difficulties which beset this subject, remains to be determined by future observation.

Physical Characters of Hemorrhage.—Having, in the preceding general view, noticed several of the more common physical characters which hemorrhage assumes, I shall now confine myself to a description of those which it presents in particular organs, viz. the brain, lungs, digestive, urinary, and genital organs, and in the skin and cellular tissue.

Brain.—When blood is effused into this organ, it is generally collected into an irregularly round mass, varying from the size of a pea to that of a hen's egg or of an orange. The blood is either of a dark red colour or almost black; coagulated, or partly fluid and partly solid. The cerebral substance in contact with the blood is always more or less ragged, and portions of it are sometimes broken down and mixed with this fluid. In some cases it possesses its natural colour and consistence; in others it is red merely from imbibition; and in others again, it is red, or red and softened from inflammation. When the blood has been washed away, the lacerated cerebral substance forming the walls of the excavation in which it was contained, presents a number of dark points or dots of coagulated blood, many of which indicate the orifices of ruptured vessels, which they close up so effectually, that some force is required to remove them by an injection before it can be made to pass into the excavation. These are the circumstances which constitute the principal physical characters of recent hemorrhage. The appearances which are observed at a subsequent period have already been described. There is one variety of cerebral hemorrhage which I may notice, as it is characteristic of the mode of operation of the cause by which it is produced, viz. mechanical violence. The blood is effused in a number of isolated portions of the grey and white substance, more

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especially of the convolutions, which, when divided with a scalpel, present a multitude of deep red dots, resembling somewhat purpura, or rather petechiæ, from the dots being small. These form groups varying from the size of a hempseed to that of an almond. The particular appearance of this kind of hemorrhage depends on the rupture of a great number of small vessels having their orifices filled with coagulated blood.

Lungs.—Pulmonary hemorrhage presents three varieties. In the first variety the blood is contained within the vesicular structure of the lung; in the second it has ruptured the air-cells and passed into the cellular tissue; and in the third there is rupture of the pleura. The situation of the blood in the first variety gives to it a peculiarity of form which does not exist in the second. In it, the effused blood, from its being enclosed within the air-cells, is collected into a round, circumscribed, solid mass, surrounded by the natural spongy tissue of the lung. The cut surface of this mass presents a deep red colour, has a homogeneous aspect, (except at the points where the bronchi and bloodvessels are situated, the open mouths of which are of a light red,) and a granular arrangement, which is partly effaced by passing the edge of a scalpel over it, thereby removing the coagulated blood from a great many of the air-cells, and producing an appearance the reverse of that which was before seen, viz. that of a minute honeycomb arrangement. The size of the masses varies from half an inch to two inches in diameter. In the second variety, the blood proceeding from the lacerated air-cells is poured into the cellular tissue, in which it generally spreads with rapidity and to a great extent, occupying sometimes the greater part of a lobe or even of a whole lung. A ragged excavation is thus formed, filled partly with fluid, partly with coagulated blood, intermixed with portions of the engorged and lacerated pulmonary tissue. The much greater extent of the effusion in this variety than in the second is owing to the blood being situated externally to the air-cells, which, being easily compressed and emptied of their contents, oppose but a slight obstacle to the progress of this fluid. The first and second varieties are sometimes observed to take place in the same portion of lung. The round form, circumscribed margin, and hardness which accompany the first, are well marked, and in the centre of this, where laceration has occurred, there is a quantity of coagulated blood. When the third variety occurs, it is as a consequence of the second, and consists in rupture of the pleura in addition to that of the cellular tissue.

When the blood is effused into the vesicular or cellular structure of the lungs, it may be retained in these situations, or a part of it may pass into the bronchi and be expectorated. The latter circumstance occurs much more frequently than the former, and the quantity of blood which takes this direction is generally in proportion to the extent of the laceration of the pulmonary tissue. In severe cases, not only are the bronchi of the affected lung, but also those of the opposite one, found filled after death with fluid and coagulated blood. In such cases the immediate cause of death is asphyxia.

The physical characters of bronchial hemorrhage do not require particular description. The blood, either fluid or coagulated, is collected chiefly in the smaller bronchi, and is seldom considerable in quantity. This latter circumstance is, no doubt, owing to the greater part of the blood being expectorated in proportion as it is exhaled from the mucous membrane; and it is probably, also, for the same reason that this fluid is not found in the vesicular structure of the lungs at the same time that it is found in the bronchi; for the rapid and extensive effusion of this fluid into the trachea from a communication being established between it and a large vessel, gives rise to pulmonary apoplexy as effectually as when the effusion originates in the vesicular structure of the

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lungs,—a fact which was first publicly noticed by Dr. Watson in his valuable lectures on hemorrhage.

I have already noticed the terminations of pulmonary hemorrhage. When a cure is effected of the first variety, (and in this variety alone has it been observed,) the granular arrangement disappears, and the circumscribed margin becomes less defined; the deep red colour passes into a dull purple or leaden hue, or assumes a lighter tint; at the same time the hardness diminishes, and the bloodvessels and bronchi which were before impermeable, permit an injection of air or water to pass through them. In proportion as the blood is removed by absorption, the natural structure of the part reappears, until at last no trace of the disease is to be observed.

Instead of being absorbed, the blood, it is said, sometimes becomes organized, or inclosed within a cyst. I have not met with an example of either.

The termination of pulmonary hemorrhage in suppuration or mortification is a very rare occurrence. I have seen only one case of the former, which was confined to a portion of the pulmonary tissue not larger than a pullet's egg, situated beneath the pleura.

Digestive Organs.—The physical characters of hemorrhage of these organs which require description are few in number, and are referable to the colour, consistence, and quantity of the effused blood. The blood effused into the stomach and intestines is seldom found to present its *natural red colour*, either when thrown out from these organs, or when contained in them after death. It has often acquired a dark purple, and still more frequently a deep brown tint resembling bistre, or the blackness of soot. The dark brown and sooty discolourations of the blood may always be regarded as the result of the action of an acid chemical agent, formed in the digestive organs, on the effused blood, except in those cases in which they are produced by the introduction of an acid poison. Hence we may conclude, that the diseases called *black vomit* and *melæna*, are mere modifications of gastric and intestinal hemorrhage, the black colour being an accidental circumstance of no importance, and derived from the chemical action of the acid product on the blood, previous to its evacuation. But as these modifications in the colour of the blood effused into the digestive organs have already been explained in the fasciculi on *Melanosis* and *Softening*, I shall not notice them further in this place.

The *consistence* of the effused blood is very generally increased with the darkness of the colour which it has acquired. It is sometimes coagulated into large masses, or into a multitude of smaller portions resembling an imperfect mixture of water, blood, and soot. This appearance is peculiarly characteristic of the action of an acid on the blood.

With regard to the *quantity* of the blood effused, it may vary from a few ounces to several pints; and although it is generally greatest in cases of perforation of an artery of the stomach, it is sometimes no less abundant when it has its source in exhalation of the mucous membrane.

Of the different local lesions which are found to accompany gastric and intestinal hemorrhage, I believe that follicular ulceration is the most frequent. The mucous membrane may be perfectly pale when the hemorrhage has proceeded from perforation of an artery; red and vascular when preceded by congestion; or it may be of a deep red colour throughout a great extent, whatever may be the source of the effusion, from imbibition alone. It almost always presents this deep red colour when the hemorrhage arises in a mechanical obstacle to the return of the venous blood, the submucous tissue being at the same time in a state of great congestion, and infiltrated with blood.

There is a peculiar form of hemorrhage of the mucous and submucous tissues of the stomach and intestines which deserves special notice, as it appears to me to be produced by the application of poisonous substances to the mucous membrane. It con-

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sists of isolated patches of a dark red, deep brown, and almost black colour, having the motley aspect of ecchymosis. The patches vary from a quarter of an inch to two or three inches in extent, and are very irregular in their form, the larger ones being apparently produced by the coalition of the smaller ones. When examined narrowly, they are found to consist either of blood alone effused into the mucous and submucous tissues, or of blood and a congeries of tortuous vessels, some of the largest of which present circumscribed dilatations where the effusion is most abundant. In this situation, also, a portion of the mucous membrane is sometimes observed in a state of sphacelus. The intervening mucous membrane may be perfectly healthy, or present a considerable degree of congestion, the tendency of which to terminate in hemorrhage is marked by the clustering together of the capillaries in numerous points, and the effusion of small specks of blood.

Urinary Organs.—Hemorrhage in these organs presents little worthy of notice as regards its physical characters, except when it occurs in the bladder. In this organ it takes place from isolated points of the mucous membrane, which, as well as its sub-cellular tissue, presents a number of deep red patches, varying from a line to half an inch in diameter, the larger ones having often a small ash-coloured slough in their centre. These patches consist of blood effused into the mucous and submucous tissues, and are accompanied by venous congestion of these tissues where the effusion has not taken place. This form of hemorrhage is chiefly observed in injuries of the spine, and appearances perfectly similar sometimes follow the application of blisters to the chest and abdomen and other parts of the body. The most frequent cause of hemorrhage from the urinary organs is the presence of the hematoid variety of cephaloma situated in the prostate, and hence its much greater frequency from these organs in the male than in the female.

Organs of Generation.—With regard to hemorrhage from these organs, I have only to notice the local lesions which have been observed to give rise to or accompany it in the unimpregnated state of the uterus. These are, congestion of the mucous membrane of the uterus; ulceration of the os tincae or of the vagina; carcinoma in these two situations; and the presence of erectile tissue in the form of polypi within the cavity of the uterus. The first of these local lesions has been occasionally observed to accompany those copious discharges of blood which occur in excessive and irregular menstruation. The second is a more obvious cause of uterine hemorrhage, and occurs most frequently as a termination of carcinoma. The third is not a frequent cause of uterine hemorrhage, but it may be the source of the most extensive periodical discharges of blood, and from its situation leaves the practitioner in utter ignorance of its nature. In one case of this kind which occurred in a married woman at the age of forty-five, the hemorrhage continued for a period of twelve years. It often took place suddenly and to a great extent, and was sometimes accompanied with the discharge of large clots of blood. The only morbid appearance observed consisted in a round flat tumour, nearly three inches in breadth and half an inch in thickness, situated at the fundus of the uterus, and projecting into the cavity of this organ in the form of a mushroom. It appeared at first sight to form part of a large fibrous tumour situated posterior to it, and contained in the substance of the uterus. It was, however, a distinct tumour, the central portion of its posterior surface being but slightly attached to the mucous membrane, and was composed of a cellulo-vascular tissue, with here and there small cavities filled with yellow-coloured serosity, or a fluid resembling chocolate. The free surface was covered by a smooth membrane, presented a mottled aspect of grey, blue, red, and yellow, and was traversed by numerous varicose vessels, some of which were pretty large. From these

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vessels, I believe, the hemorrhage proceeded, and it is probable that the periodical character of the discharge and the frequency of its occurrence depended on the erectile nature of the tumour.

The ovaries are sometimes the seat of hemorrhage. In several cases which I have observed, the blood was effused within the capsules of the ova, which were distended to the size of a large garden pea, and occurred in young plethoric females. One of them died after a violent hysterical paroxysm, and not only were the capsules of the ova distended with blood, but the vessels of the fallopian tubes and broad ligaments were in a state of extreme congestion, and nearly a table-spoonful of blood was thrown out on the mucous surface of the uterus.

Skin and Cellular Tissue.—The only forms of hemorrhage to which I shall allude as occurring in the cutaneous and cellular tissues, are those which constitute what are called petechiæ, purpura, and scorbutic blotches or ecchymoses. The differences observed in the physical characters of these three forms of hemorrhage, are referable chiefly to the extent, form, and situation of the effused blood, although it must be remarked that all three may occur in succession in the same individual. When the hemorrhage gives rise to the formation of *petechiæ*, the blood is collected in minute isolated points, situated immediately beneath the cuticle, presenting sometimes a light, at other times a deep red or purple colour, and varying in size from the fourth of a line to a line in diameter. An increase of the hemorrhage and the formation of larger circular spots, varying from one line to four or six lines in diameter, sometimes of a light, but much more frequently of a deep red, purple, livid, or almost black colour, having their seat between the rete mucosum and cuticle, in the cutis and subjacent cellular tissue, constitute *purpura*, of which there are two important varieties, *purpura simplex* and *purpura hemorrhagica*. The lighter colour of the blood, the smaller size of the spots, and their diffusion over a less extent of surface in the former than in the latter, are the physical characters which may sometimes be observed as marking a difference between the two. But the essential difference, and that to which any importance is to be attached, consists in the effusion being confined to the skin and cellular tissue in *purpura simplex*; and its occurrence not only in these situations, but in the mucous and submucous tissues of the respiratory, digestive, urinary, and generative organs, and in the subserous cellular tissue of various parts of the body in *purpura hemorrhagica*. The physical characters of the hemorrhage which occurs in *scorbutus* differ in general sufficiently from those of *purpura*, to enable us to distinguish the one from the other. The effused blood in *scorbutus* gives rise to the formation of blotches varying from the breadth of a shilling to that of the hand, situated on various parts of the body, but most frequently and extensively on the inferior extremities. Their colour and general appearance give them a striking resemblance to the ecchymosis which follows a severe bruise, and in patients affected with this disease, can indeed be produced even by slight pressure, particularly in those parts of the body where the skin is thin and the cellular tissue abundant. When the blood is extensively infiltrated in the cellular tissue, a considerable degree of hardness accompanies the discolouration; and when rupture follows the effusion, and the blood accumulates beneath the skin, a soft spongy sensation is communicated to the touch. In proportion as the blood is removed by absorption, the dull red, purple, and black colour of the blotches assume a brown, green, and yellow colour in succession, and ultimately disappear. These colours indicating the disparition of the old and the formation of new blotches are observed at the same time, in *scorbutus* and *purpura*, and modify considerably the general appearance of both.

DESCRIPTION OF THE PLATES.

PLATE I.

Fig. 1. Cerebral apoplexy ; AA, external surface of the brain; BB, left lateral ventricle laid open ; C, thalamus ; D, corpus striatum ; E, a large effusion consisting of fluid and coagulated blood, protruding into the ventricle through an extensive laceration of the thalamus ; F, large congested vessels opening into the hemorrhagic excavation. *Fig. 2,* hemorrhage in the same situation ; AA, external surface of the brain ; B, corpus callosum divided longitudinally ; C, corpus striatum ; D, thalamus ; E, the lacerated surface of this body ; F and G, layers of the fibrous structure of the brain separated by the effused blood, and presenting deep red points in the situation of the ruptured vessels ; H, a quantity of coagulated blood in the fourth ventricle and commencement of the spinal canal. *Fig. 3,* a longitudinal section of the pons Varolii and medulla oblongata, representing hemorrhage of the former, and rupture of the inferior wall of the fourth ventricle. A, the pons ; B, portion of the cerebellum ; C, fourth ventricle ; D, vertebral and basilar arteries ; E, the hemorrhage. *Fig. 4,* the same part of the brain cut transversely, representing hemorrhage in the direction of its fibrous structure. A, the pons ; B, medulla oblongata ; C, cerebellum ; D, the effused blood. *Fig. 5* is a representation of that form of hemorrhage produced by a blow on the head. AA, the convolutions of a portion of one of the hemispheres of the brain, from which the membranes have been removed ; B, C, D, the isolated hemorrhagic patches of various sizes, in the largest of which the mouths of the ruptured vessels are distinguished by black points. *Fig. 6* represents a vertical section of a large patch ; A, the white substance of the brain, into which the effused blood has penetrated ; B, the grey substance in which the effusion generally originates ; C, the effusion. *Fig. 7,* hemorrhage of the membranes of the brain. A, left half of the dura mater and arachnoid ; BB, falx ; C, longitudinal sinus ; DDD, the arachnoid, a portion of which is turned aside to shew the effused blood G, lying between it and the dura mater F, from which it is separated by a false membrane EE.

PLATE II.

Figs. 1, 2, and 3, represent the cure of cerebral hemorrhage. *Fig. 1 ;* A, lateral ventricle ; B, brown substance of corpus striatum ; C, the cellulo-vascular tissue, of a straw-yellow colour, occupying the excavation formed by the effused blood, which has entirely disappeared, except in two or three points D, where a very small quantity, of a reddish-brown colour, still remains. A small hemorrhagic excavation in the same stage of cure is seen at E. *Fig. 2* is a section of a portion of the corpus striatum A, and thalamus B ; the latter containing an apoplectic cyst C, lined by a serous membrane D. In *fig. 3* the organization of the fibrine of the blood has not given rise to the formation of a serous cyst. The fibrine is collected into a solid mass, having a fibrous

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arrangement. It occupies the whole of the excavation, and is united to the contiguous cerebral substance by minute bloodvessels. A, pons Varolii; B, basilar artery; C, the organized fibrinous substance. *Fig. 4*, hemorrhage of the vesicular structure of the lungs; AA, cut surface of the pulmonary tissue; B, a section of the circumscribed effusion which characterises this form of pulmonary hemorrhage; C, the divided extremities of the bronchi and bloodvessels, recognised by the lighter red colour of their parietes, from the deep red granular aspect of the general mass; D, the honeycomb appearance produced by passing the edge of a scalpel over the surface of the section, and removing the coagulated blood from the distended air-cells. *Fig. 5* represents the second and third varieties of pulmonary hemorrhage; AA, the healthy pulmonary tissue; B, the hemorrhage terminating in the former by an irregular diffuse margin; C, D, laceration of the pulmonary tissue; E, the pleura detached and raised by the effused blood, and perforated at F and G. *Fig. 6* represents the appearances observed in the pulmonary and subserous tissue in purpura hemorrhagica. A, substance of the lung containing several hemorrhagic spots B and C of various sizes; and others DD, situated beneath the pleura. *Fig. 7*, a section A, of a portion of lung; B, the appearance presented by vesicular hemorrhage after the blood has been partly absorbed. The same form of pulmonary hemorrhage is seen at C, terminating in suppuration, principally of the interlobular cellular tissue D, and accompanied by slight inflammation E, of the substance of the lung.

P L A T E I I I .

Fig. 1, hemorrhage of the mucous membrane of the stomach from a mechanical obstacle to the return of the blood, situated in the heart. A, pyloric portion of stomach; B, commencement of duodenum; C, the mucous membrane thrown into large folds, of a uniform red colour, and presenting a granular appearance from enlargement of its follicles. *Fig. 2*, hemorrhage of the stomach from follicular ulceration. AA, enlarged follicles, the bodies of which are pale, but have a red central orifice, the situation of the ulceration or erosion, and source of the hemorrhage; B, other enlarged follicles having also the red central point, but differing from the former in their bodies being red; C, enlarged follicles, pale throughout, but having a central depression or orifice, probably the morbid state in which they exist before the occurrence of hemorrhage. D, petechiæ of the mucous membrane, which sometimes accompany this form of hemorrhage. *Fig. 3*, hemorrhage of the duodenum from ulceration of the mucous membrane. AA, duodenum; B, pyloric portion of the stomach; CCC, ragged ulcers of the mucous membrane, penetrating to the submucous tissue, and surrounded by a red elevated border of the former. The surface of the ulcers is covered by dark coagulated blood, and the mucous membrane presents a deep brownish red colour from imbibition. D, a small ulcer apparently situated in one of the mucous follicles; EE, two small ulcers in the stomach, having a similar situation, and presenting in their centre a yellow-coloured slough. *Fig. 4*, hemorrhage from perforation of the coronary artery of the stomach. A, œsophagus; B, trunk of the coronary artery; C, an ulcer nearly cicatrized situated over the perforated artery, the orifice of which contained a fibrinous coagulum D, having in its centre a small opening through which the blood escaped, and is indicated in the plate by the probe E. Several other ulcers had existed in the stomach, but were completely cicatrized, as seen at F and G. *Fig. 5*, pulmonary hemorrhage from perforation of a large branch of the pulmonary artery and a contiguous bronchial tube, from tuber-

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culous ulceration. AA, vomicae; BB, tubercles; C, trachea; D, a large irregular ulcer situated in the left bronchus, near the bifurcation of the trachea, and penetrating into the pulmonary artery E, and forming a direct communication F, between these two tubes, through which the blood escaped so abundantly as to prove fatal in less than a quarter of an hour. *Fig. 6* represents the hemorrhagic congestion of the mucous and submucous tissues of the digestive organs, produced by certain poisonous substances. At A the hemorrhagic patches present a uniform dull red colour with slight congestion only, the effused blood being confined to the mucous tissue; at B and C the effusion is more extensive, occupies the submucous as well as the mucous tissue, and is accompanied by great venous congestion and a varicose distribution of the vessels, presenting here and there circumscribed dilations surrounded by deep red or black coagula. Sometimes the blood is poured chiefly into the submucous tissue D, where it accumulates and raises considerably the mucous membrane.

P L A T E I V .

Fig. 1. Hemorrhagic congestion of the urinary bladder. AA, lateral lobes of the prostate; B, enlarged middle lobe of this gland; CC, patches of blood of various sizes, effused into the mucous and submucous tissues, several of them presenting a yellow slough in their centre. *Fig. 2* represents an erectile tumour of the uterus which gave rise to frequent and extensive hemorrhage. A, portion of vagina; B, cavity of uterus greatly enlarged; C, a fibrous tumour lodged in the substance of the uterus and projecting inwards, covered by the mucous membrane D; E, the erectile tumour rising above the surface of the uterus, covered by a smooth glossy membrane, and traversed by a multitude of varicose vessels, from which the hemorrhage proceeded. *Figs. 3 and 4,* cutaneous and subcutaneous hemorrhage. *Fig. 3,* A, the spots of purpura hemorrhagica; B, those of purpura simplex; C, petechiae. *Fig. 4,* the spots and blotches of scorbutus. A large blotch is represented at A, in the upper part of which are seen the various shades of colour which the skin exhibits when the blood is recently effused; whilst those which succeed the gradual absorption of this fluid are observed in the inferior part at B. There is also represented at C a variety of the scorbutic hemorrhage, which presents a considerable resemblance in point of form to purpura hemorrhagica. *Fig. 5* represents the morbid anatomy of the parts which give rise to hemorrhoids. There are two forms of this disease, the more common of which depends on dilatation of the veins of the rectum; the other on a transformation of the dense cellular tissue of the margin of the anus into erectile tissue. A and B represent this tissue in the progress of transformation. It forms a pendulous tumour, at first chiefly composed of cellulo-fibrous tissue A, which afterwards acquires a cellulo-vascular structure, the vessels being grouped together into small isolated masses, separated by fibrous tissue, as seen at B; at last it acquires a spongy structure filled with blood, and then resembles a section of the spleen or spongy body of the penis. It is in this state that hemorrhage is most apt to occur. C indicates other tumours in the cellulo-fibrous state. The dilatation of the veins which terminates in hemorrhage is sometimes very conspicuous, as in the case represented in this plate. The tumour D is composed of the dilated extremities, probably of several veins, has a large cellular structure filled with dark coagulated and fluid blood, and communicates with several veins E, in a varicose state, also distended with blood. These veins again communicate freely with others, FF, some of which, G, are large and tortuous.

Plate 1.



Plate II.

Fig 1

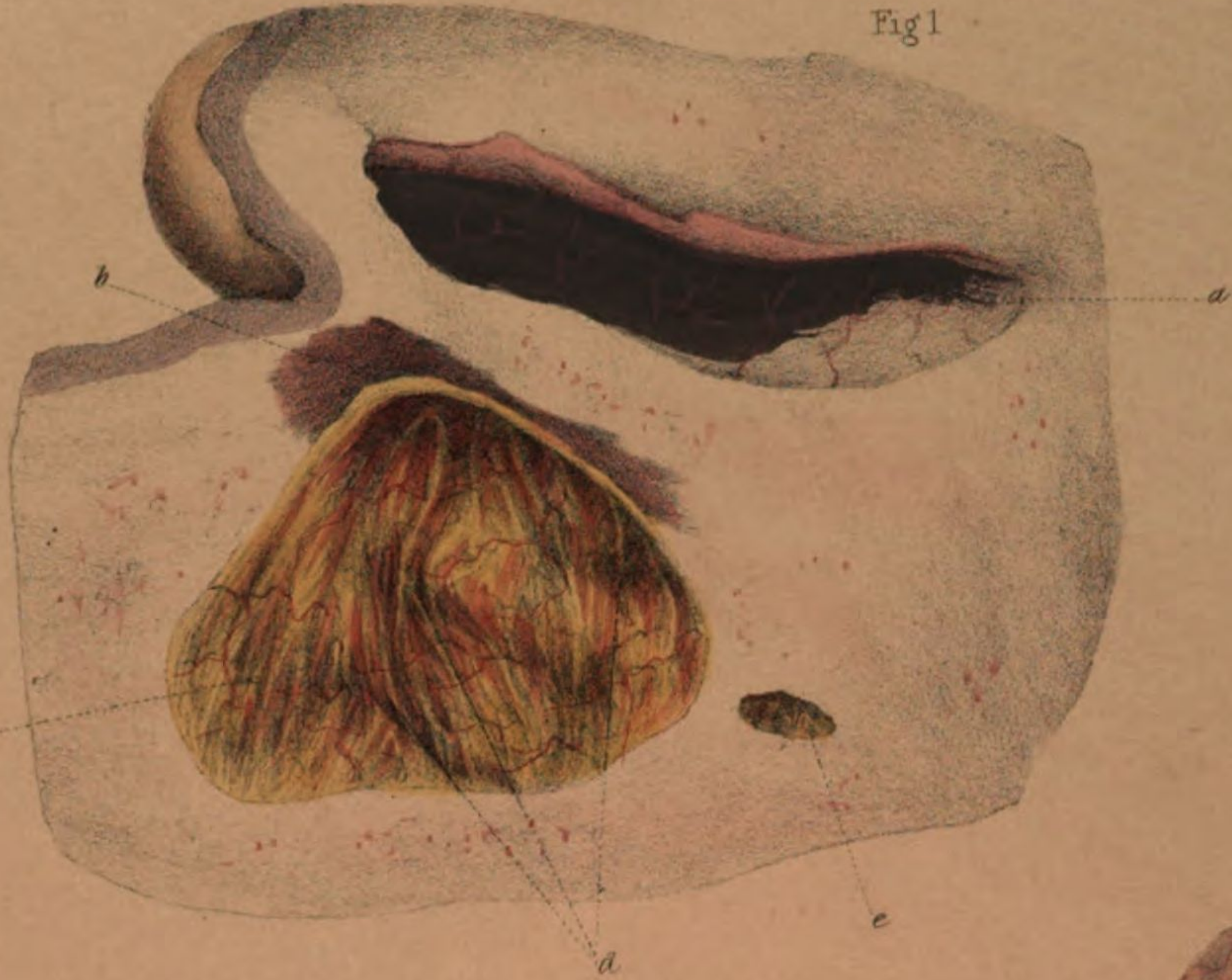


Fig 3.



Fig 2.



Fig 7.

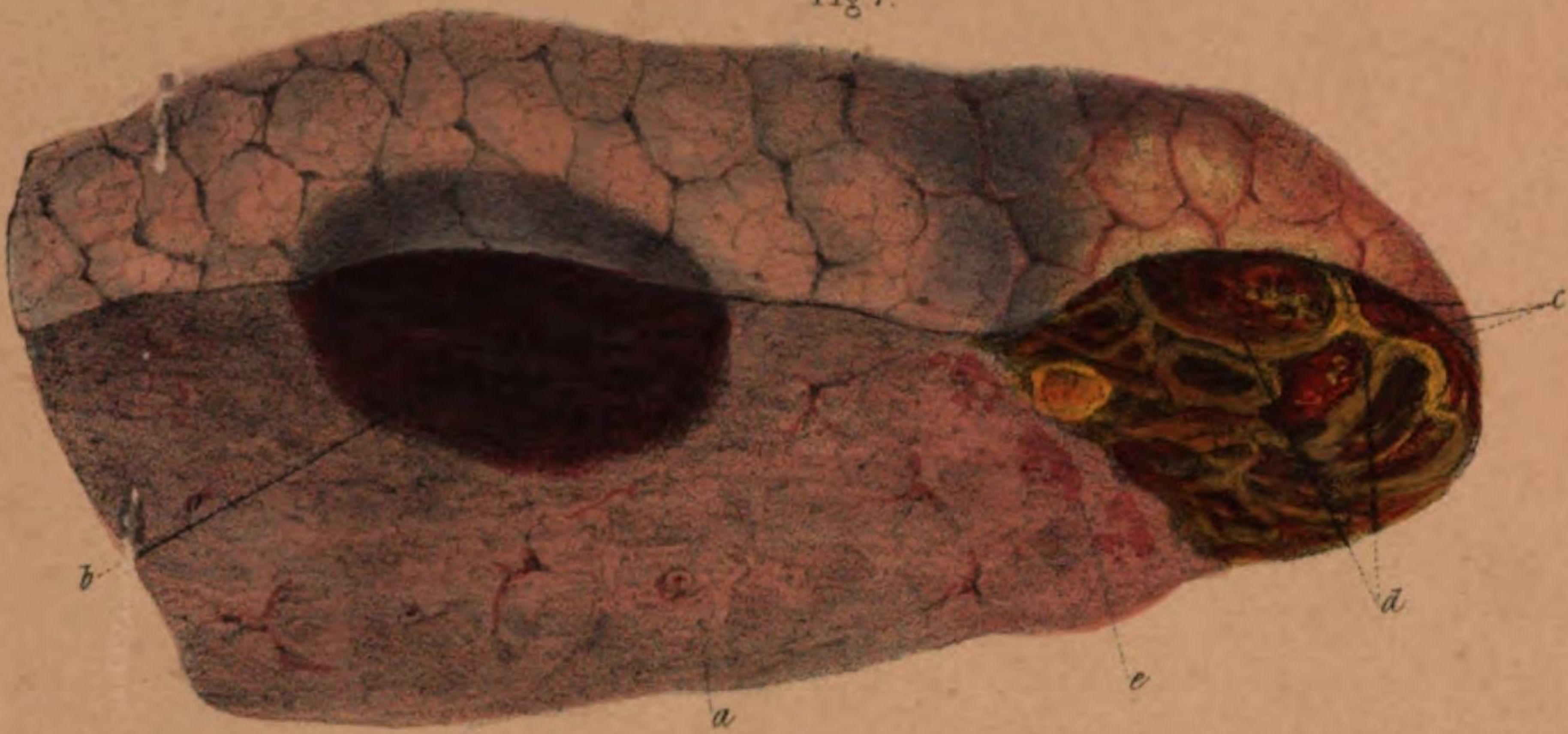


Fig 6.

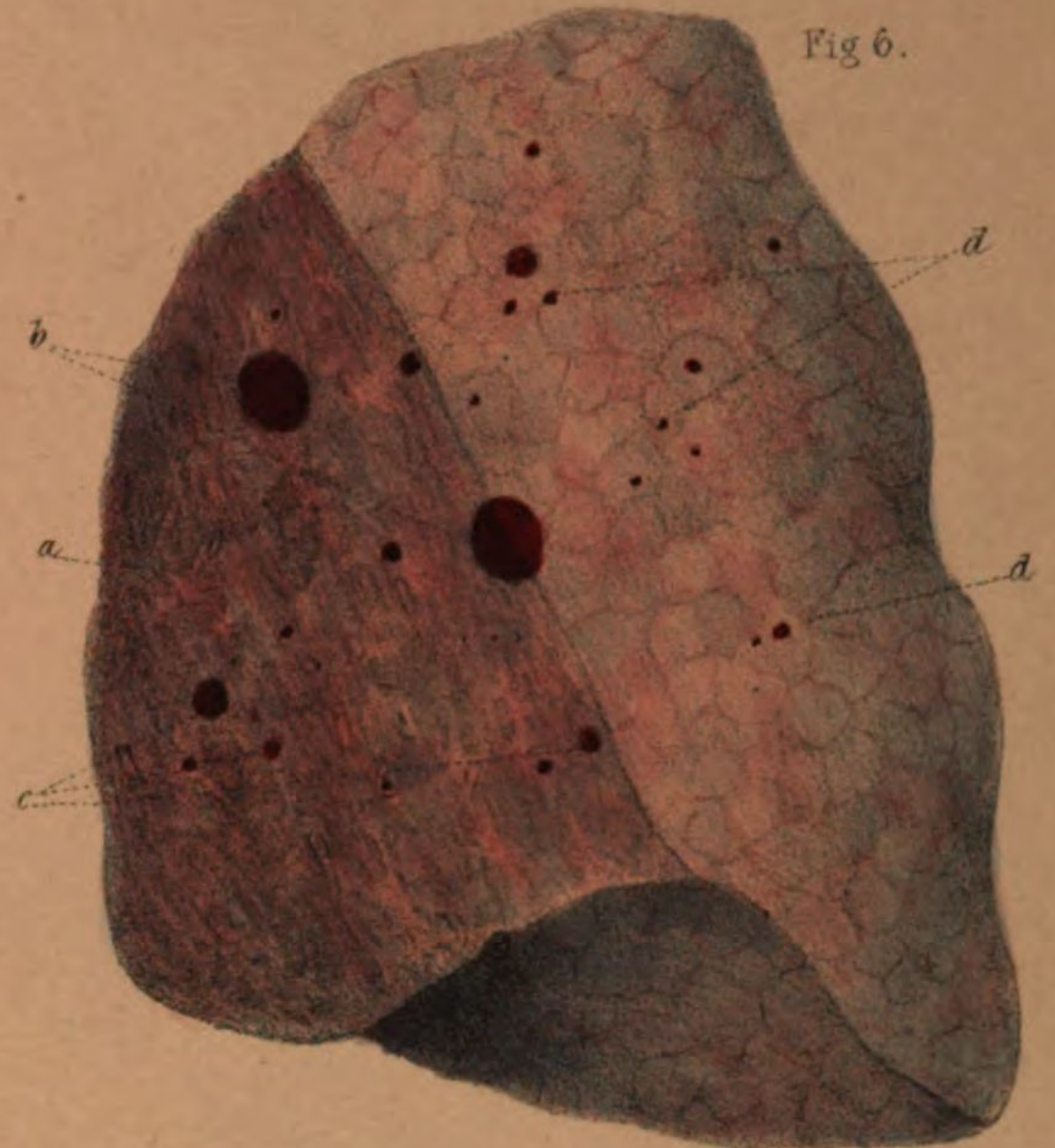


Fig 4.



Fig 5

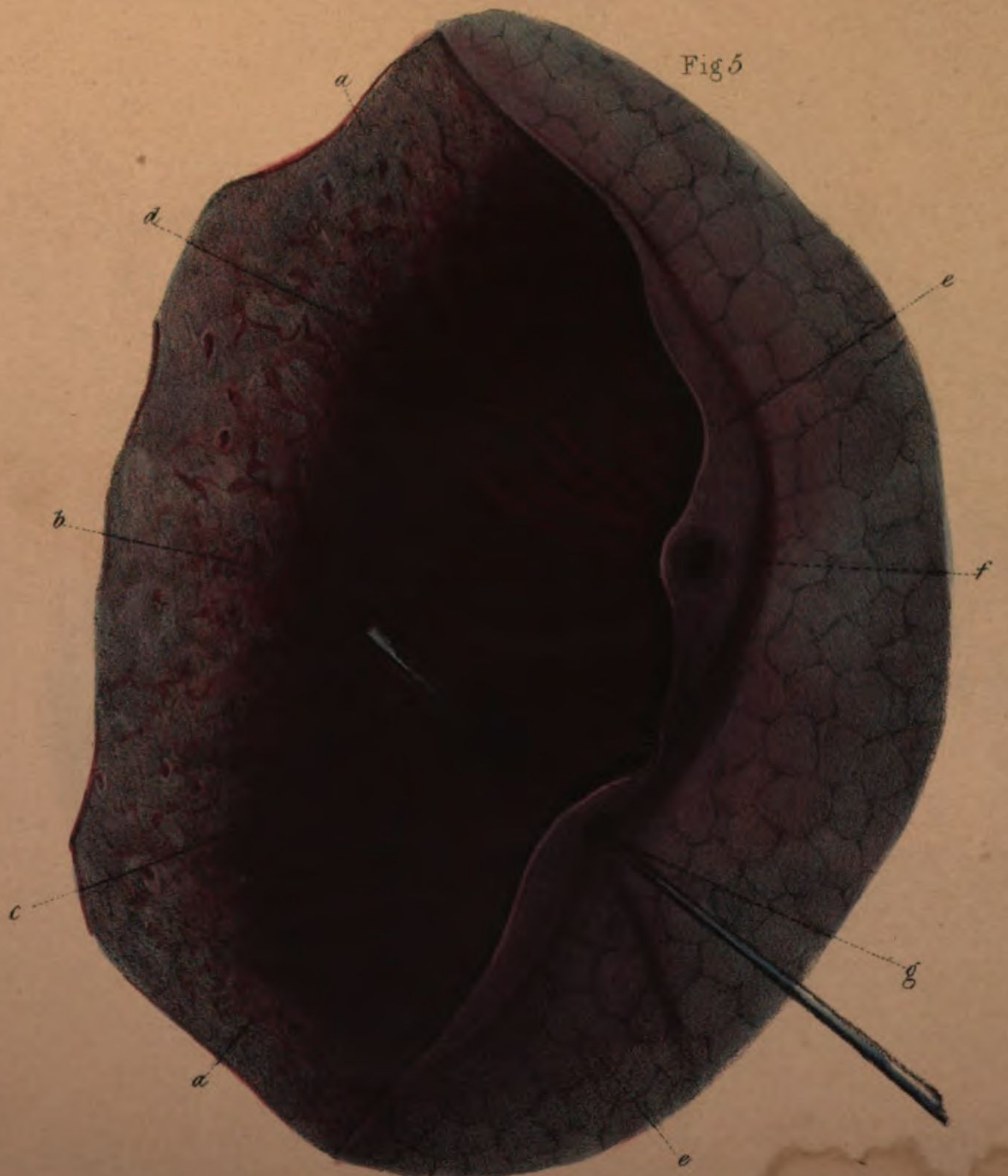


Plate III.

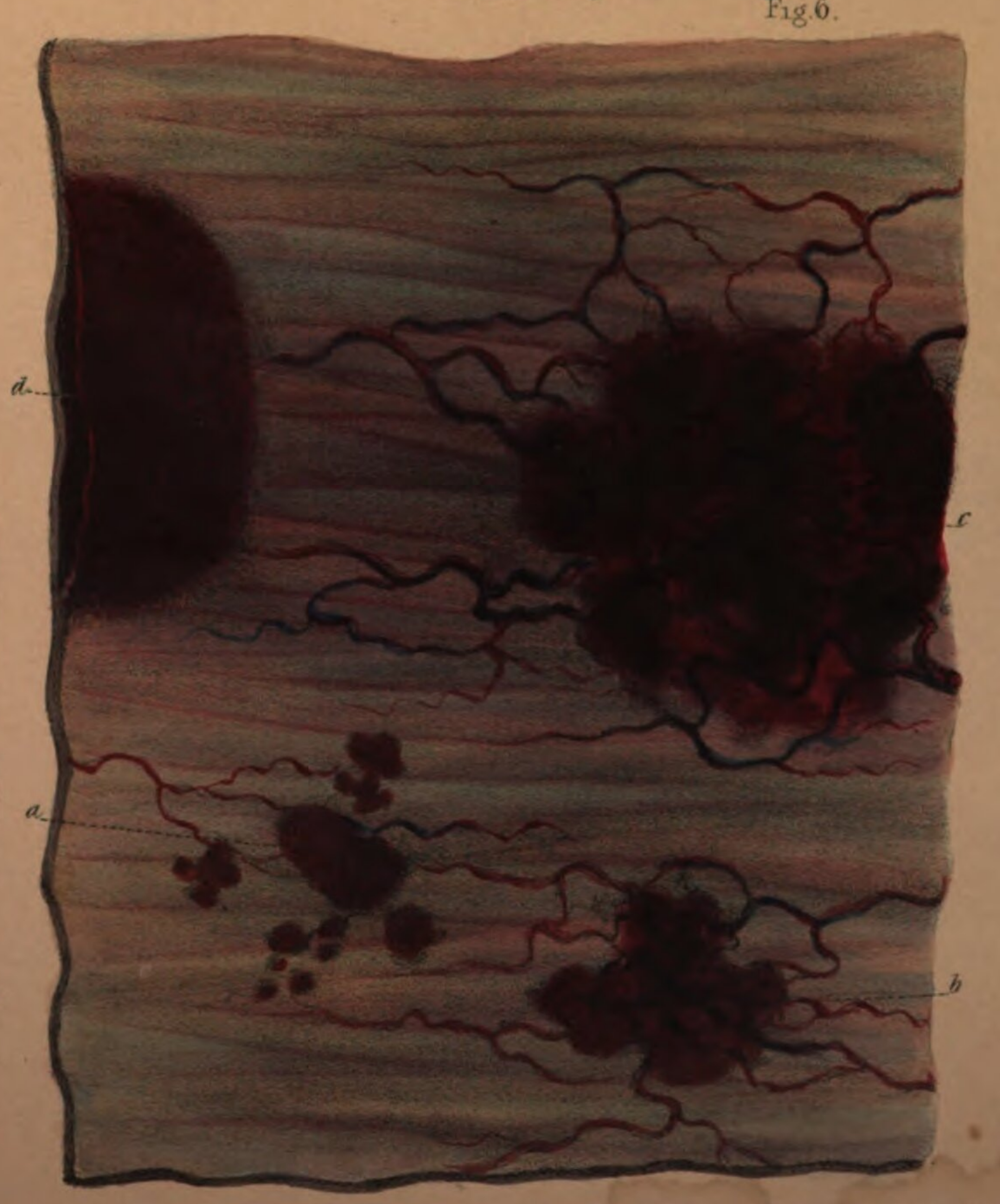
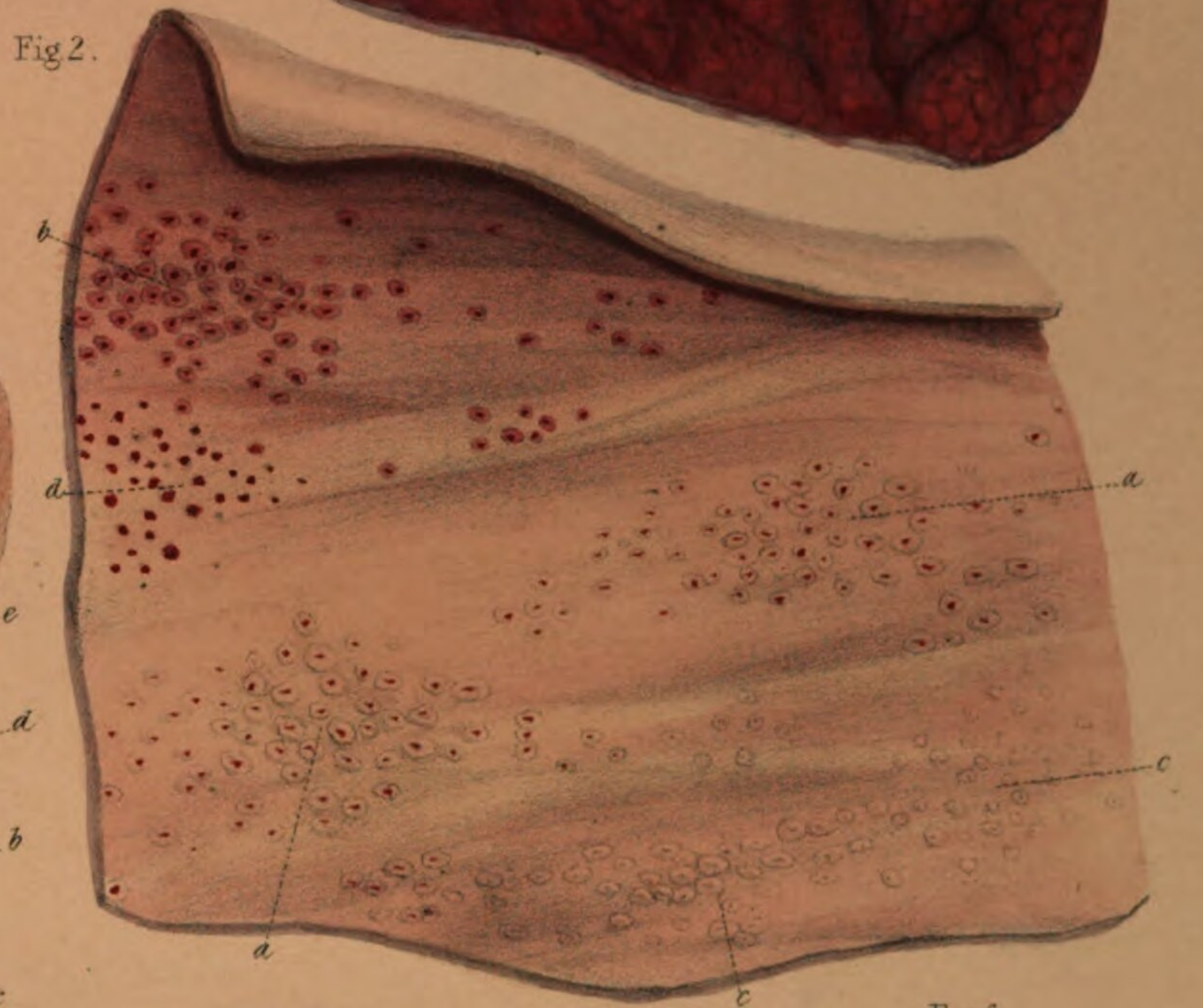
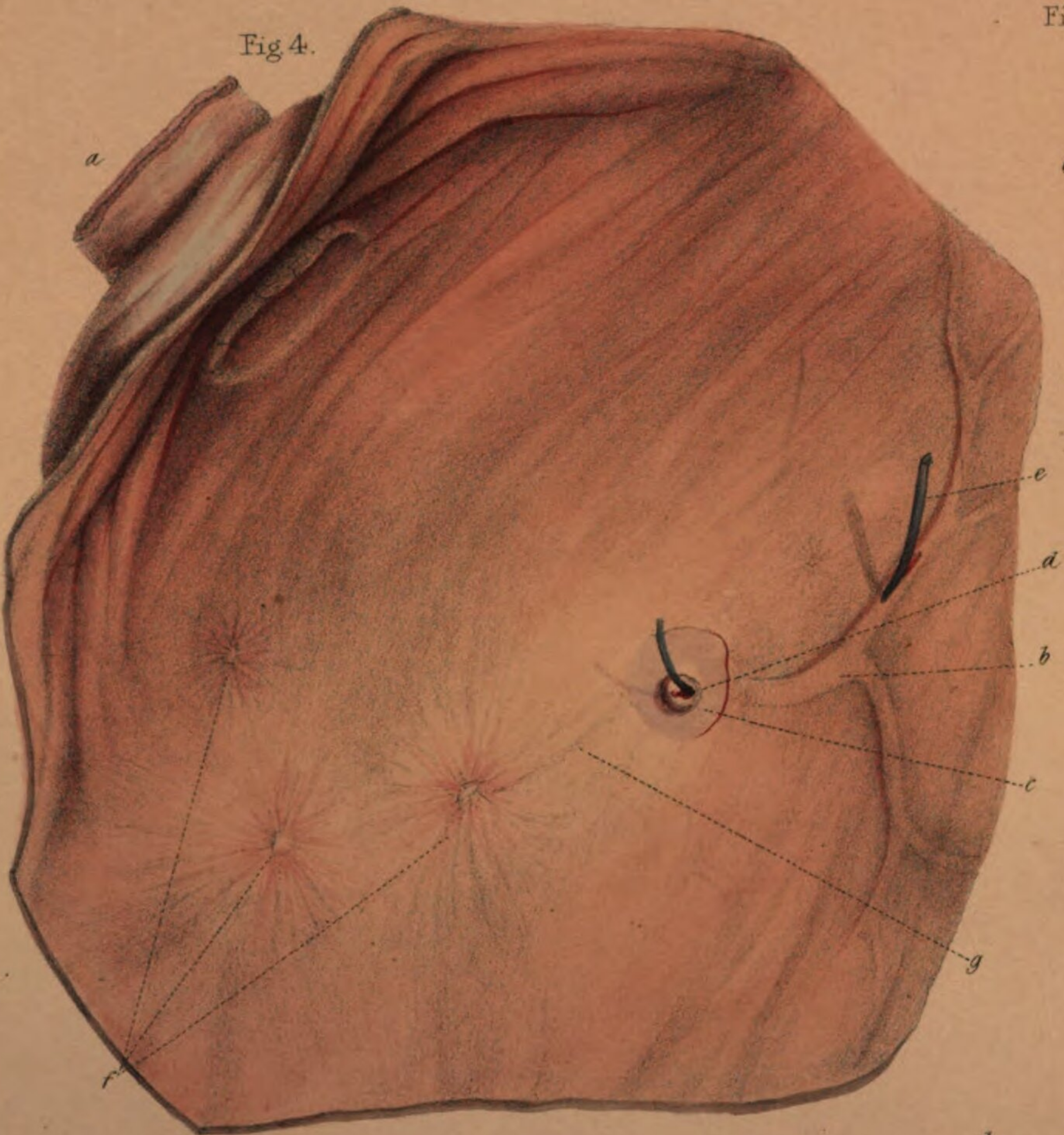
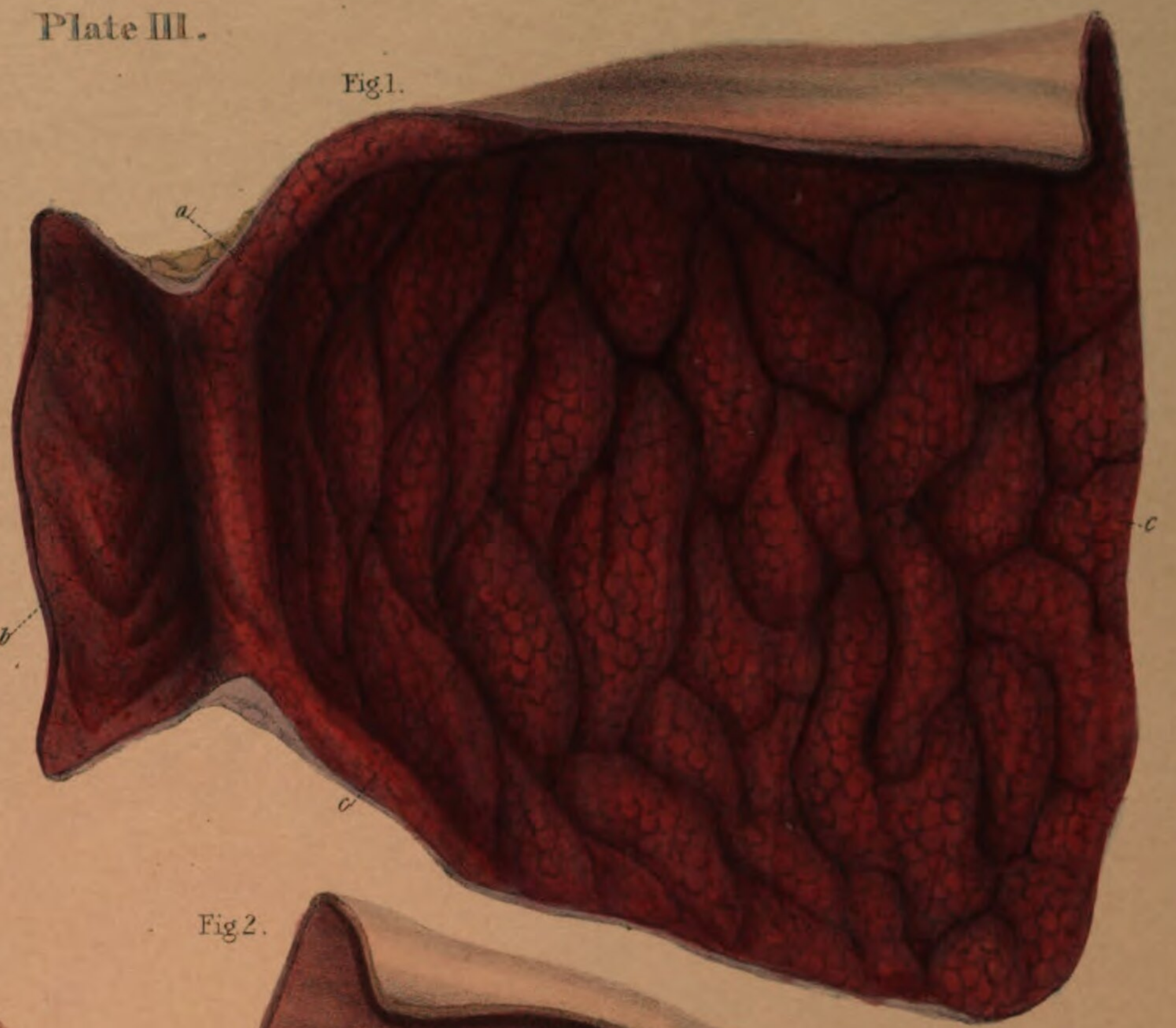
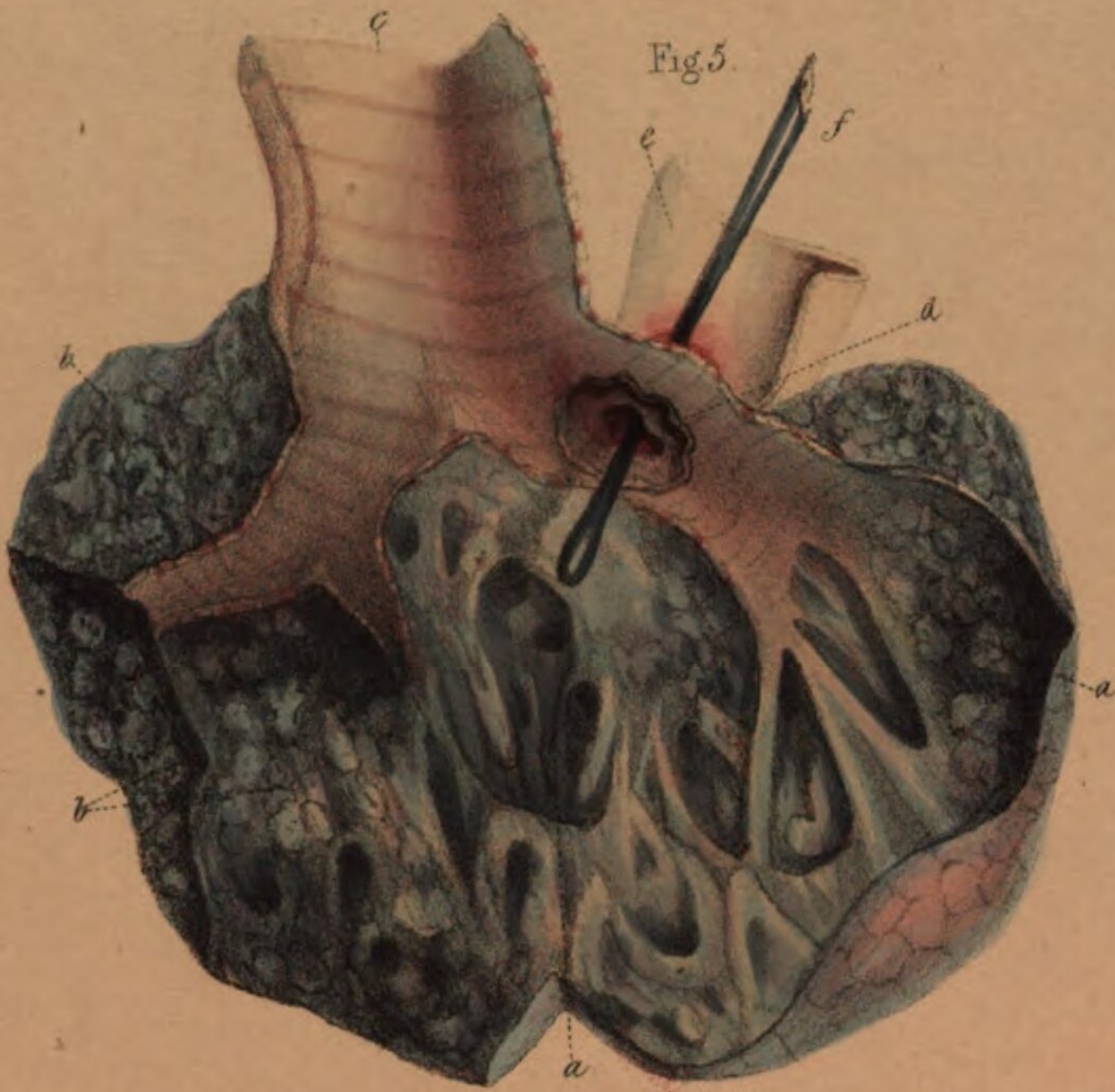
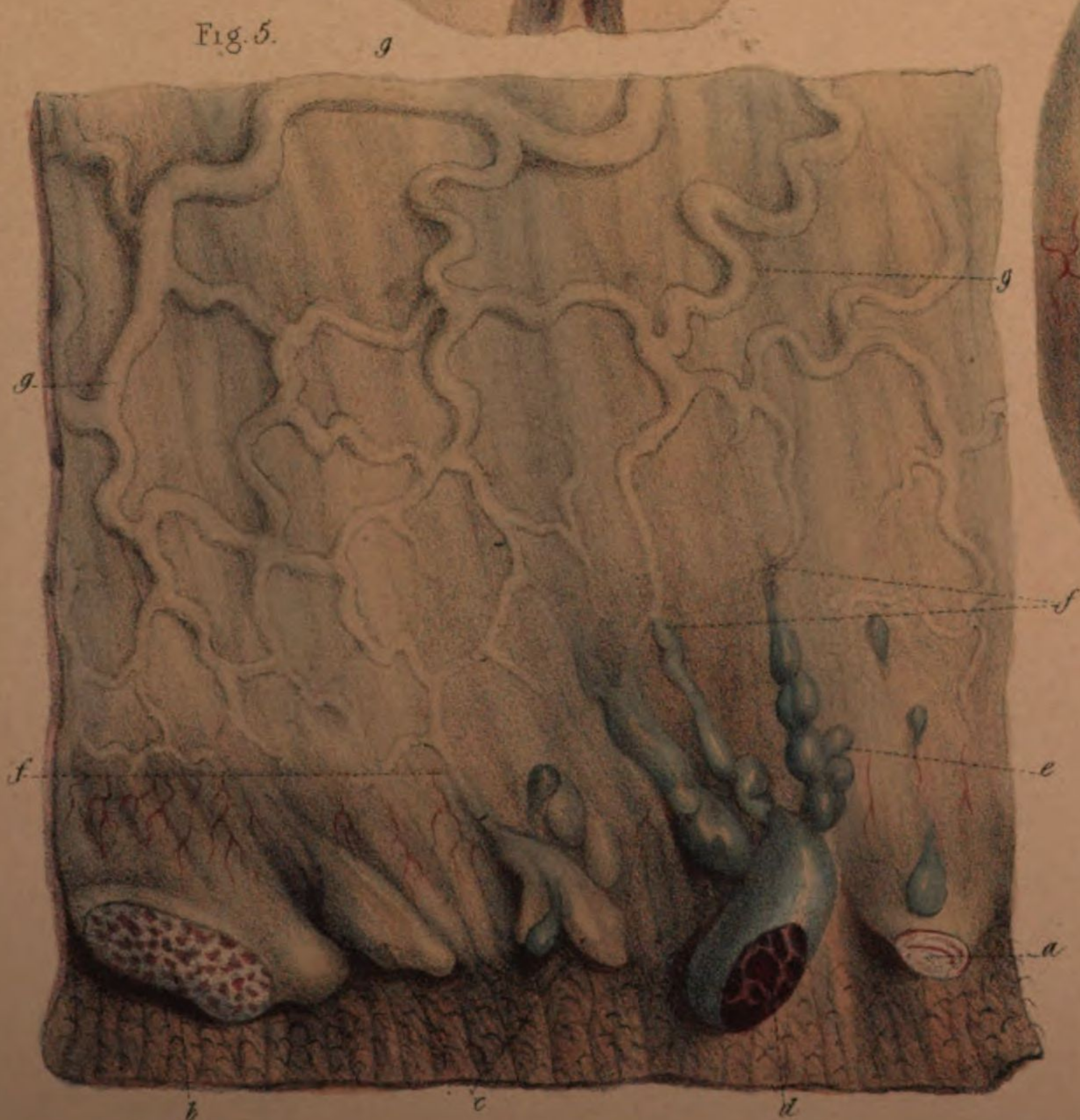
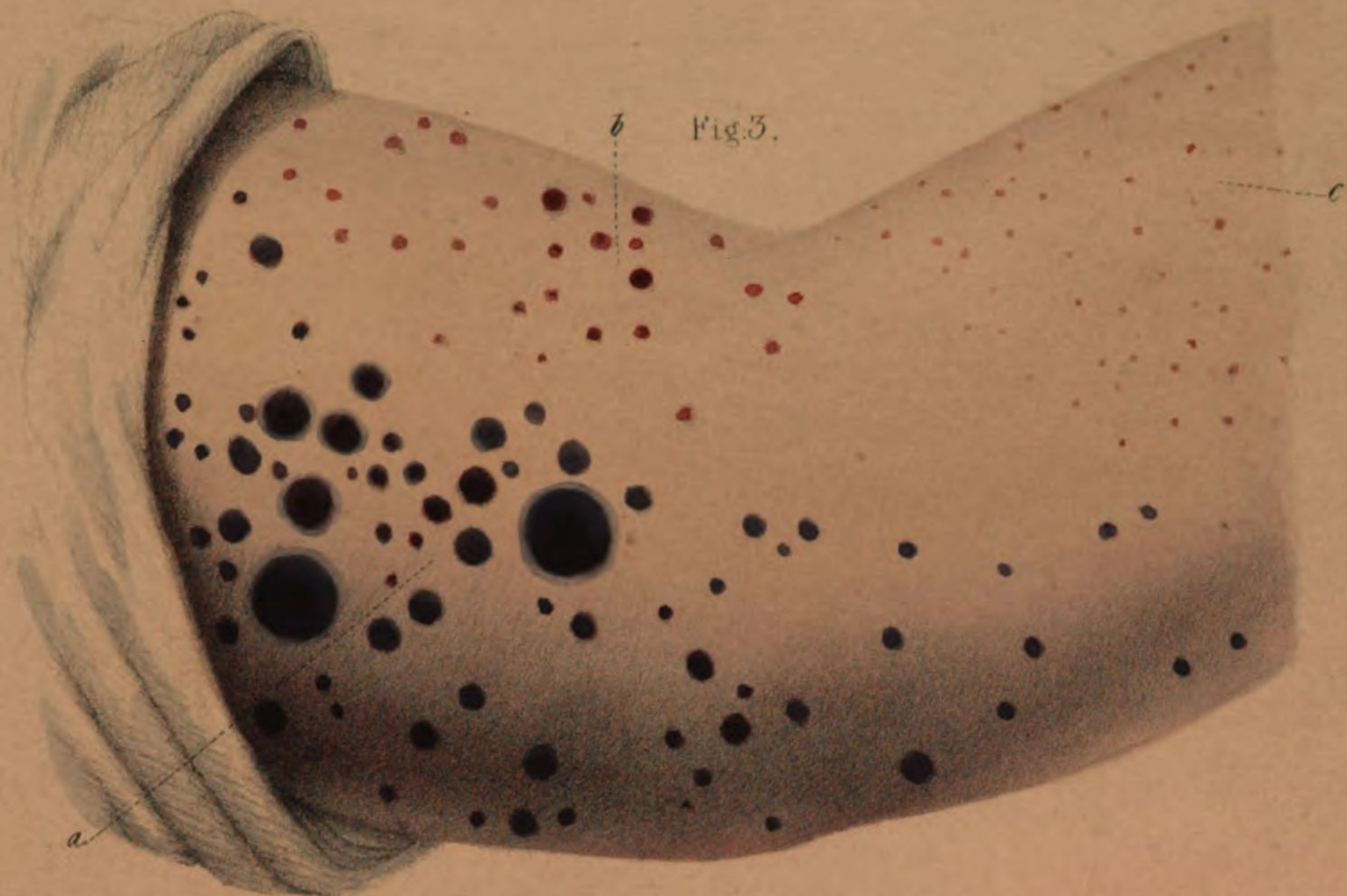


Plate IV.



Drawn on Stone by Dr. Carsonell.

Drawn by Haghe Esch. to the King. 17 Gale 53.

SOFTENING.

THE term *Softening* is employed to designate a diminution of the natural and healthy consistence of organs. It is only of late years that this lesion, denominated by French pathologists *Ramollissement*, has been investigated and described as a special morbid condition; and whether we consider the frequency of its occurrence, the variety which it presents as to degree and extent, the serious and often fatal effects to which it gives rise, or the wide difference of its nature in the same or in different organs, it constitutes a subject of great interest and importance.

Before proceeding to describe the physical characters of softening, it may be proper to allude to those circumstances under the immediate influence of which this change of consistence appears to take place.

The pathological phenomena which precede softening are of various kinds. Those of inflammation are by far the most frequent. There is, indeed, no organ in which softening may not occur as the mediate or immediate consequence of inflammation. Obliteration of the arteries produced by accidental products contained within these vessels is likewise a pathological condition which gives rise to softening. Of these two kinds of softening, the first seldom affects many organs or tissues at the same time in the same individual; and the second is confined to the brain. But there is another kind of this lesion of a much more general character, and which is also very different in its nature from the two former. It occurs in almost all the textures of the body at the same time, although it may be so slight in some as hardly to be observable; whilst in others, even the hardest, it may be strongly marked. It is never observed, unless in individuals in whom nutrition in general is greatly modified. The modification of nutrition which precedes the softening process is, however, very different in kind in different individuals—a difference which obviously exercises a great influence in determining the seat and severity of the disease. Thus, in children born in a state of debility and emaciation, and in those who have long been deprived of the wholesome necessities of life, we find all the tissues and organs of the body more or less soft, and easily injured by external causes. This general diminution of cohesion is accompanied by universal pallor, a watery, scanty, and aplastic state of the blood. Such, also, is the case in advanced stages of tuberculous disease and scorbutus; the bones as well as the other textures being found, in those who die of these diseases, soft, spongy, and infiltrated with a sero-albuminous or sero-sanguinolent fluid. In another class of patients, the softening, while it pervades to a certain extent all the tissues of the body, exists in a much greater degree in the bones, and from the supercumbent weight which they have to support, or the impulse which they receive from the action of their muscles, they lose their natural forms, and become bent or flattened to an extraordinary degree. There is likewise a modification of nutrition which is accompanied by softening of

[*Softening*, 1.]

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all the tissues, and which seems to depend on a morbid condition of the blood. The softening is not conspicuous till after death, and is always in proportion to the fluidity of the blood and the tendency which this fluid has manifested to run into putrefaction. Such is the case in the worst forms of typhoid fevers, small-pox, scarlatina, measles, &c., and when certain poisons have been introduced into the circulation.

There is a variety of softening, or rather flaccidity, which requires to be noticed. It is best seen in the skin and cellular tissue when these textures, after having been greatly distended by an accumulation of fluid in the abdomen, are left unsupported by the removal of the distending cause. The looseness or flaccidity of these tissues is also very conspicuous in old people; in persons who, from a state of obesity, become lean, and in persons in general who become rapidly emaciated, particularly from disease.

Besides these pathological conditions of the consistence of tissues and organs, there are others which do not occur until after death. Two of these, arising from maceration and putrefaction, require only to be mentioned. The third, produced by the action of the gastric juice on the stomach and intestines, deserves particular notice, in consequence of its giving rise to appearances which have often deceived the practical pathologist.

From these general remarks the several kinds of softening may be arranged as follows:—

I. SOFTENING OCCURRING DURING LIFE.

1. *From Inflammation.*
2. *From Obliteration of Arteries.*
3. *From Modifications of Nutrition.*

II. SOFTENING OCCURRING AFTER DEATH.

1. *From the Chemical Action of the Gastric Juice.*
2. *From Maceration and Putrefaction.*

I shall, at present, endeavour to illustrate three only of these five kinds of softening, viz. softening from inflammation, from obliteration of arteries, and the chemical action of the gastric juice.

I. *Softening from Inflammation.*—It may be laid down as a general rule, that every organ or tissue affected with acute inflammation, undergoes at the same time a diminution of consistence. The principle on which this change is effected is obvious, viz. cessation of the circulation, and, consequently, of nutrition, in the inflamed tissue. This state of the circulation is generally admitted to constitute one of the essential local characters of inflammation. It occurs subsequently to the accumulation of the blood in the capillaries, is always preceded by the effusion of serosity, and, in general, is accompanied by the formation and deposition of coagulable lymph and pus. Under these circumstances a supply of assimilable materials is cut off, and the function of nutrition is entirely suspended. A diminution in the consistence of the tissue thus situated follows, the degree of which will vary with the duration of the suspended function, and the degree of cohesion possessed by the tissue in its normal condition.

The process of softening appears to be accomplished under the immediate influence of two agents, a mechanical agent on the one hand, and a vital agent on the other. Thus, the effused fluids separate, mechanically, the molecules of the tissue which the cessation of nutrition had deprived of their vital properties; or in other words, the cessation of nutrition deprives the molecules of those properties on which their power of

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aggregation depends, and in this state they are separated and detached by the effused fluids.

It is important to observe that the cessation of circulation is accompanied by that of absorption; for were this not the case, the affected tissue, instead of being converted into a soft or pulpy mass, would be partially or wholly removed, and a solution of continuity formed, such, in fact, as we find to be the case after the inflammation has disappeared, and circulation with its dependent function, absorption, has been restored.

Physical Characters of Inflammatory Softening.—From the preceding general outline of the changes which take place in a tissue affected with inflammatory softening, it must be obvious that the physical characters of this lesion will present considerable variety. They will vary, as has already been remarked, not only with the duration of the cause on which the softening depends, and the natural difference of consistence of the tissue in which it takes place, but also with the structure of the affected organ, and the quantity of blood which it contains.

The physical characters of inflammatory softening consist principally in the degree and extent of this change, and the modifications of colour, form, and bulk, by which it is accompanied. As this kind of softening is no where so conspicuous as in the brain, I shall commence with the description of its physical characters, such as they are observed in this organ.

1. *Inflammatory Softening of the Brain.*—The degree of softening of the cerebral substance may vary from a slight diminution of the natural consistence of the part affected, to that of cream or milk. The first stage of softening of this substance is often so slight that it is hardly perceptible to the touch, and may, even when considerable, if not accompanied by some peculiarity of colour, be altogether overlooked. In the first case a gentle stream of water allowed to fall upon the cerebral substance is the best means of ascertaining whether a portion of it has undergone a diminution of its natural consistence; and in the second case, the only way of detecting the presence of softening unaccompanied by any obvious change of form or colour, is to submit the whole of the cerebral substance to a careful inspection, by removing it piecemeal in the form of thin slices.

In the first stage the cerebral substance is not yet broken down; it has only lost a certain degree of its cohesion, for it is still continuous with that by which it is surrounded. In the second stage the diminution of consistence is so great as to be recognised at first sight, owing to the change of *form* by which it is accompanied. The cerebral substance sinks by its own weight beneath the level of the cut surface; and prominent parts, such as the thalami, corpora striata, and convolutions, become more or less flattened. In the third stage a solution of continuity has been effected by the separation and partial removal of the softened cerebral substance. It is now of the consistence of cream or milk, contained in an excavation of variable extent, situated in the substance of the brain, or confined between the membranes and convolutions of this organ. All these degrees of softening may be met with in the same or different portions of the brain at the same time.

Softening varies greatly in *extent*. It may occur in the brown or white substance of any part of the brain; in one portion alone, or in several portions at the same time, as the septum lucidum, fornix, and walls of the lateral ventricles; the corpora striata and thalami; a portion of one or both hemispheres; the brain and cerebellum; and is rarely met with in the latter organ without being present in the former. In all

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these parts of the brain, the softening may be confined to a very limited spot, or pervade their entire substance. There are examples of nearly the whole of one of the hemispheres of the brain in adults having undergone this change of consistence, and both hemispheres have been found reduced to a pulpy or almost fluid consistence in children, probably on account of the natural softness of this organ at an early period of life.

The *colour* of inflammatory softening of the cerebral substance presents considerable variety. The principal varieties of colour depend on the quantity of blood contained in the affected part, in changes which this fluid undergoes some time after its accumulation or effusion, and on the presence of serosity or pus. Redness and vascularity are, in general, greater in the first than in the second stage of softening, but the degree and the extent of either greatly depend on the quantity of blood in the cerebral vascular system. In some cases the redness and vascularity extend to a considerable distance beyond the softened part, diminishing gradually in intensity; in others they are limited to the immediate vicinity of the softening. The vascularity of the softened cerebral substance may be ramiform or punctiform, but it has more frequently a hemorrhagic character. When this substance is divided, it presents a number of red points, streaks, or patches, produced by the blood accumulated in the veins, or by the effusion of this fluid. In some cases the effused blood is small in quantity compared with the extent of the softening; in others it pervades the whole of the softened substance, and sometimes presents the appearance of hemorrhagic apoplexy. The redness, vascularity, and hemorrhagic character of inflammatory softening, are seldom so conspicuous as when this lesion occupies the brown substance, more especially of the corpora striata and thalami.

Inflammatory softening of the cerebral substance is not always accompanied by those changes of colour which I have just described. The softened substance sometimes preserves its natural colour, or it may be even paler than natural. Thus the septum lucidum may be converted into a mere pulp, without its colour being perceptibly altered; and the same degree of softening may take place in the brown substance, while at the same time it is so pale as hardly to be distinguished from the white or medullary in its vicinity. Pale softening in either of these situations, viz. in the white and brown substance of the brain, is a frequent occurrence in hydrocephalus, and it is also this variety of softening which is sometimes met with in those fevers in which the brain is primarily or secondarily affected. In such cases the substance of the brain in general is pale; its vascular system contains but a small quantity of blood, and its membranes are infiltrated with serosity. It is, in fact, to the pressure which the effused serosity exercises on the bloodvessels, that the anæmic condition of the brain in general, and of the softened portion of it in particular, is to be attributed. When the pale softening is the consequence of meningitis, it is often confined to the brown substance of the convolutions, and is frequently detected while removing the pia mater, in consequence of portions of this substance being carried away with the bloodvessels which connect it with this membrane.

When the redness which accompanies softening arises from the presence of effused blood, it may always be regarded as evidence that the softening is of recent occurrence. But there are other modifications of colour which accompany softening of the cerebral substance, and which indicate that the disease has existed for some time—several weeks, two or three months. The principal modifications of this kind consist of brown, yellow, and orange colours, either separate or combined, and which occupy either the softened substance, the part of the brain contiguous to it, or both at the same time. They

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are not observed unless the softening has been accompanied by sanguineous effusion, and originate in changes taking place in the effused blood. Such are the modifications of colour to which I allude, so frequently observed to take place in the blood effused into the subcutaneous cellular tissue in consequence of external violence. The brown colour appears first, and is very limited in extent compared with the orange and yellow by which it is succeeded,—circumstances which enable us to form a tolerably accurate opinion regarding the extent of the sanguineous effusion by which the softening had been accompanied.

A pale yellow straw-coloured tinge of the softened cerebral substance arises also from the presence of pus. But this is rarely observed unless the softened substance be in contact with the membranes of the brain. The presence of serosity produces, as I have already said, the pale softening; but it likewise communicates a glossy albuminous aspect to softening, which it does not present in any other circumstances.

When softening of the cerebral substance is accompanied by an increase of bulk, which is seldom considerable, it is chiefly owing to the presence of serosity.

Such is a description of the physical characters of inflammatory softening of the brain, which applies to the same lesion of the cerebellum, spinal cord, and nerves.

2. *Inflammatory Softening of the Mucous Membranes.*—Softening of the mucous membranes in general is a very frequent occurrence, but it is much more so in some than in others, and most of all in the digestive mucous membrane. Indeed, the extreme frequency of this lesion, as well as the extent to which it proceeds, particularly in the mucous membrane of the stomach, has excited in an especial manner, and most deservedly, the attention of pathologists. Soon after the promulgation of the *Doctrines Physiologiques*, there was a general tendency, more especially in France, to ascribe the origin of all, or of the greater number of diseases, to inflammation or some modification of this pathological state, the physical characters of which were sought for with equal zeal by those who believed and disbelieved in these doctrines. Softening being one of these characters of inflammation, its presence alone was regarded as sufficient evidence of the previous existence of inflammation; the absence of redness or vascularity not being considered a valid objection to the inflammatory origin of this alteration, additional importance was attached to this change of consistence, in consequence of the frequency of its occurrence in the stomach and intestines being brought forward as strong evidence in favour of the statement, viz. that the local cause of Fever consists in inflammation of the mucous membrane of these organs. In order, therefore, to arrive at a satisfactory solution of several of the most important questions in pathology, it became absolutely necessary to determine the nature of softening of the mucous membrane of the stomach and intestines; whether and how far this lesion is to be attributed to inflammation or some other local cause, and what are the characters by means of which either or both are to be recognised. The description which I am about to give of softening of the mucous membrane will show that this lesion is produced under circumstances of the most opposite kind, viz. during life in consequence of inflammation, and after death from the chemical action of the gastric juice. I shall describe the first kind only of softening in this place; that of the second kind will be given under the head of the general division of our subject to which it belongs.

Physical Characters of Softening of the Mucous Membrane of the Stomach and Intestines.—Softening of the mucous membrane of these organs presents, in regard to degree and extent, the same variety as in the brain. In the first stage the mucous membrane, instead of possessing that degree of cohesion peculiar to it in different

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portions of the alimentary canal, and which permits of its being detached from the submucous tissue in pieces of considerable size, breaks when seized between the fingers or forceps; in the second stage, the edge of a scalpel, or even the finger passed lightly over its surface, converts it into a soft, somewhat opaque, creamy-looking pulp; and in the third stage it is so completely softened and detached, that a gentle stream of water poured on it from the height of a few inches removes it entirely from the surface of the submucous tissue. The removal of portions of the mucous membrane of greater or less extent is effected in a similar manner during life, by the contents of the stomach and intestines in their passage through these organs. It is important to observe that in all these stages the softened mucous membrane is more or less *opaque*; it does not present the *transparency* which accompanies softening from the chemical action of the gastric juice. In the former case, and in the last stage of softening, it resembles a mixture of flour and cold water or milk; in the latter, the same materials after having been submitted to the action of heat.

The *extent* of inflammatory softening of the mucous membrane is extremely variable. It may be limited to a small portion, or comprehend nearly the whole of this membrane. It has been described as extending from the mucous membrane to the other tunics of the stomach and intestines, and terminating in perforation of these organs. Such, however, does not appear to me ever to be the result of inflammatory softening. It is the consequence of the chemical action of the gastric juice.

The greater *frequency* of inflammatory softening in some portions than in others of the digestive mucous membrane, is a circumstance which requires to be particularly noticed, because it is a means of enabling us to distinguish this disease from mere chemical dissolution. It is said that softening of this membrane occurs more frequently in the stomach than in the intestines, a statement which is true in so far only as regards the occurrence of this lesion without any reference to its nature. It is not true as regards inflammatory softening, while it is true if applied to softening as a chemical effect of the gastric juice. Nor is it a fact that inflammatory softening, or any other pathological modification of this lesion, is most frequent in that portion of the mucous membrane which lines the fundus of the stomach; for it is precisely at the fundus of the stomach that the mucous membrane is almost always acted upon by the gastric juice after death. In the stomach inflammatory softening of the mucous membrane occurs most frequently in the body and pyloric portion of this organ; in the intestines, at the termination of the ileum, in the depending and fixed portions of the colon, as in the cœcum, the right and left hypochondriac regions and sigmoid flexures of this intestine.

Lastly, inflammatory softening is more frequent in the follicular than in the villous structure of the mucous membrane. It is from this latter circumstance in particular that the *form* of inflammatory softening derives any diagnostic value. When the softening affects the glandulæ agminatæ and solitariae, it presents the general form of these glands, viz. large oval, or small circular patches, and these are forms of softening which are always to be regarded as the result of a pathological cause, particularly if the surrounding mucous membrane does not present the same change, or any of the other physical characters of inflammation. Softening of the mucous membrane of the stomach, in the form of stripes and bands, has been described with great care and precision by Mons. Louis, and has been much insisted upon as characteristic of inflammatory softening; but to which, for reasons I shall afterwards assign, I am disposed to ascribe a very different origin.

The *colour* of the softened mucous membrane varies extremely, but the principal

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varieties of colour are the red and pale. In the *red* softening the redness may be confined to the softened part of the membrane, or it may extend to the other parts at the same time; or the latter may be red and the former pale. The redness may also be very slight or very considerable, and presents no peculiarity of form. In the *pale* softening, the mucous membrane presents a pale greyish, or yellowish grey tint, its natural colour being little altered; or it may be paler than natural, when it generally presents a milky aspect, owing to its being reduced to a thin layer, through which the colour of the submucous tissue is partially seen.

With regard to the physical characters of inflammatory softening of the mucous membrane in other organs, as the respiratory, urinary, and generative, nothing requires to be said, as they differ chiefly in degree from those which accompany this lesion in the digestive organs, and more especially as they do not indicate the existence of any other modification of consistence than that which is produced by inflammation. It may, however, be observed that softening is seldom considerable in the mucous membrane of these organs, except in that of the uterus, larynx, and bronchi; and is least of all in that of the urinary organs. It derives no peculiarity of form in any of them, unless when it affects the follicular structure of the mucous membrane of the trachea or bronchi, or of the vagina: but this is very seldom observed.

3. *Inflammatory Softening of the Cellular Tissue.*—Inflammatory softening of the cellular tissue is, in several respects, a very important pathological lesion. This tissue is by far the most frequent seat of softening, and it is in consequence of its having undergone this change, that parenchymatous organs in general, when affected with inflammation, are easily lacerated and broken down. This is well exemplified in pneumonia; the diminution of cohesion of this tissue, whether inter-lobular or inter-vesicular, leaves the other anatomical elements as it were without their natural connecting medium, so that little resistance is opposed to any mechanical means employed to separate them. The facility with which this separation is effected, is at the same time favoured by the presence of the effused fluids, especially in the vesicular structure of the lung.

Muscular tissue is also easily torn or separated into shreds, in consequence of softening of the interstitial cellular tissue by which its fibres are united. This is often observed in the muscles of voluntary motion, and sometimes in the heart. The easy separation of the serous and mucous membranes is always the consequence of inflammatory softening of their connecting cellular tissue, and the degree of facility with which their separation is effected, affords a ready means of estimating the degree and extent of the inflammation to which this tissue had been subjected. Cases of peritonitis and meningitis occur which would escape our *post-mortem* researches but for this state of softening of the cellular tissue. In such cases there may be little or no increase of vascularity, and perhaps only a slight serous effusion, which may be overlooked, or if observed, can afford no idea of the degree and extent of this morbid condition of the cellular tissue. Both are readily ascertained in peritonitis, by making a circular incision of the peritoneum, and then pulling the intestine in a longitudinal direction. The intestine is thus as it were unsheathed, by the gradual separation of its muscular coat from the peritoneum. In meningitis the bloodvessels are separated from the pia mater in a similar manner.

The other physical characters of inflammatory softening of the cellular tissue do not require to be noticed. With regard to the physical characters of this lesion in other tissues, it is only necessary to observe that they vary chiefly in degree from those already described.

II. *Softening from Obliteration of Arteries.*—Softening from obliteration of arteries

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occurs only in the brain and at an advanced period of life. It is this kind of softening which was first described by M. Rostan as a disease *sui generis*, as entirely opposite in its nature to inflammation, and which he likened to *gangrena senilis*. The opinion of this author met with strong opposition from many pathologists, but more especially from Lallemand of Montpellier, who maintains that softening of the brain is always the consequence of inflammation. This latter opinion, which is by far the most generally received one, is as far from the truth as the former is ambiguous and inconclusive. Indeed, no pathologist who has investigated this subject has, so far as I know, furnished us with evidence that the real nature of this kind of softening, to which the brain is subject in the aged, has been ascertained. It has been conjectured to originate in ossification of the arteries, yet even M. Rostan, among the great number of cases of softening of the brain, the histories of which are detailed in his work, has not given a single case in which ossification and obliteration of the arteries of this organ are mentioned as having been observed at the autopsy.

Physical Characters of Softening of the Brain from Obliteration of the Arteries of this Organ.—The most essential of these characters is the state of obliteration of the arteries; for it is only by the obvious existence of this state that we can distinguish this kind of softening from that which is the consequence of inflammation. The obliteration of the arteries may depend on the presence of fibrous, cartilaginous, or osseous substances, formed in the interior of these vessels or between their coats. These accidental products may exist in the form of cylinders occupying the entire caliber of arteries of considerable size, and also the smaller branches; or they may form patches or small masses projecting internally, which obstruct the circulation of the blood. The cessation of the circulation in the diseased arteries probably takes place gradually, and a supply of the materials of nutrition being ultimately cut off from the portion of the brain to which these arteries are distributed, softening follows in the manner which has already been explained.

The obliterated arteries may occupy the softened cerebral substance, and can be seen ramifying through it; and when this substance is removed by pouring water upon it, the solidified arteries retain their situation, and feel sometimes to the touch as hard as fine wires. If the obliteration be confined to a limited portion of an artery whose branches terminate in the softened part of the brain, the cause of the softening may be overlooked. In the case of obliteration of minute arteries, or of a single small arterial trunk, the softening is generally confined to a space not exceeding an inch or two inches in superficial extent; but if several large contiguous branches be obliterated, the extent of the softening is considerably increased, or occupies two or more distinct portions of the brain; and if the obliteration has taken place in the carotid or one of its principal divisions within the cranium, the greater part or the whole of a hemisphere may be softened.

This kind of softening is, like the inflammatory, not confined to any particular portion of the brain. Like the latter, it occurs also more frequently in the brown than in the white cerebral substance, or in those parts most abundantly supplied with blood-vessels, as in the optic thalami, corpora striata, and brown substance of the convolutions. Softening from obliteration of the corpus callosum, septum lucidum, and fornix is extremely rare. I have met with only one example of it in the pons Varolii, which is represented in *fig. 4. Plate IV.*; once also in the cerebellum, but never in the spinal cord.

The degree of softening from obliteration, as well as the various colours which this

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change presents, are often very similar to those observed in inflammatory softening. It is necessary, however, to observe that redness is seldom considerable, and that vascularity and effusion of blood are generally wanting on account of the impervious state of the arteries. When effusion takes place, it is probably the consequence of rupture of the obliterated vessels, or of some of the smaller ones having remained pervious and yielding to the increased momentum of the blood.

3. *Softening from the Chemical Action of the Gastric Juice.*

This lesion, which, for obvious reasons, occurs essentially in the digestive organs, was first observed by John Hunter, while engaged in a series of experiments on digestion. He described it as *digestion of the walls of the stomach after death*, and as the immediate consequence of the solvent property of the healthy gastric juice. Although the opinions of Hunter on this subject were confirmed by the subsequent observations and experiments of Spallanzani, Dr. Adams, Mr. Allan Burns, and others, it was not till very lately that their importance was fully appreciated by the application of the principle which they embraced, to the pathology of the digestive organs. British pathologists appear to have overlooked the importance of the subject in relation to those morbid conditions of the stomach which bear a resemblance to the *post-mortem* effects of the gastric juice upon this organ. In France the opinions of Hunter were at first received with doubt, afterwards turned into ridicule, and, instead of their having been submitted to the test of experiment, the most vague and absurd hypotheses were set up in their place by Chaussier, Morin, Laisné, and many others. The researches of Jæger and several other pathologists in Germany, and of Dr. John Gardner in this country, seemed only to have revived the original opinions of Hunter, without removing the objections which had been urged against their validity; for the *post-mortem* effects of the gastric juice were continually or almost daily described in France as pathological lesions. The memoir of M. Cruveilhier on the “*Ramollissement Gélatiniforme, &c.*,” and that of M. Louis on the “*Ramollissement avec Amincissement, et de la Destruction de la Membrane Muqueuse de l’Estomac*,” furnish striking examples of this kind; for as regards the local lesions which these two distinguished pathologists have described, they are, in almost every instance, the *post-mortem* effects of the gastric juice.

I regret that want of space will not permit me to notice the various opinions which have been given of this kind of softening of the stomach and intestines, described also under the appellations of erosion and perforation of these organs; and I regret more that, for the same reason, I am prevented from giving even a summary of the numerous experiments which I have made on healthy animals, to arrive at an accurate knowledge of the *post-mortem* effects of the gastric juice. The results of these experiments agree in all respects with those of my observations on softening, erosion, and perforation of the digestive organs in the human subject, and shew that these lesions, under all their forms, and such as they have been described as the consequence of disease, are produced after death by the dissolvent property of the gastric juice. It is necessary to observe that the essential character of the gastric juice is *acidity*. Without this property (which is the immediate agent of digestion), there is no dissolution of the stomach, intestine, or other organs after death. In every case of this lesion which I have observed, this property of the gastric juice was present, and was readily detected by its sour smell on laying open the stomach, or by means of litmus paper. That this, and no other, is the chemical cause of dissolution, is put beyond all doubt by *neutralizing* the gastric juice in the stomach of a healthy animal, killed during the act of digestion; for when this is done, *dissolution does not take place*. This, therefore, being the cause of the

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lesions I am about to describe, I shall use, in preference to the term gastric juice, that of gastric acid.

Physical Characters of Softening, Erosion, and Perforation of the Stomach, &c. from the Action of the Gastric Acid after Death.

Situation.—The situation of softening, erosion, and perforation, is a circumstance of great importance, for as these lesions almost always occur, or are greatest in degree in the most depending portion of the stomach, viz. the fundus, it at once leads us to suspect that a physical agent is concerned in their production. It is, in fact, owing to the gastric acid or digested food gravitating towards the fundus or any other portion of the stomach which has accidentally acquired a depending position, that the particular locality of these lesions is determined. Their occurrence in any other portion of the stomach is an exception to the rule, and may be explained by the presence of certain physical conditions of this organ. Thus, great enlargement of the spleen may elevate or compress the fundus of the stomach so as to prevent the gastric acid from collecting within it, while at the same time this fluid accumulates in some other portion of the stomach, as the pyloric, which may have become the most depending part; great distention of the transverse arch of the colon may throw the great curvature of the stomach upwards and forwards, when the fluid contents of this organ collect along the small curvature; or tumours in the liver, retro-peritoneal cellular tissue, &c. may be so situated as to produce considerable variation in the position and form of the stomach, and, consequently, in the situation in which the effects of the gastric acid are observed. Besides, the situation of the chemical dissolution of the stomach will vary with the position given to the body after death.

Degree.—The degree of this kind of softening varies from a slight diminution of consistence of the coats of the stomach, to their conversion into a gelatinous pulp. In the first stage the mucous membrane (which is that which is first dissolved,) presents a slight diminution of consistence, and has acquired a certain degree of transparency. When seized, it breaks immediately, or is crushed between the fingers into a soft pulp. In the second stage this membrane is seen lying like a quantity of albumen covering the submucous coat, and can be wiped off or removed by a bit of cloth or a stream of water. In the last stage the mucous membrane has entirely disappeared to a certain extent, thus leaving its submucous coat denuded, which is recognized by its grey silvery aspect. The same degrees of softening are observed in the other tunics of the stomach.

Extent.—Chemical dissolution of the stomach may be confined to a small portion of the fundus, or occupy nearly the whole of this organ. It may be limited to the mucous membrane or extend to all the other tunics. In the latter case perforation of the stomach is effected, the gastric acid escapes, is brought in contact with the contiguous organs, as the intestines, liver, spleen, and diaphragm; dissolves portions of the one and perforates the other; passes into the cavity of the chest and produces similar effects. The quantity of the gastric acid relative to the bulk of the stomach, modifies considerably the extent of chemical dissolution. It is when this fluid is in great abundance, or the stomach distended with gas, that the cardiac orifice becomes dilated, and the gastric acid passes into the œsophagus, the cuticular lining, mucous and muscular coats of which it dissolves, and forms a communication between the cavity of this tube and that of the chest.

Form.—The form of chemical softening of the coats of the stomach by the gastric acid presents several important varieties. If the softening be confined to the mucous

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membrane of the fundus, the form which it assumes is that of small or large patches. These are generally irregular, their borders being formed by the mucous membrane, and the bottoms of each by the submucous coat; their edges, besides being irregular, are thin, soft, and somewhat transparent. If the softening has extended to the other coats of the stomach, the edges of these are beveled outwards, present a fringed appearance, or terminate in thin irregular prolongations, which, when water is poured upon them, are seen to float like shreds of transparent coagulable lymph. Such are the forms of softening of the mucous membrane, so long as this membrane is smooth or stretched out by the contents of the stomach. But when this membrane is thrown into folds, or forms plicæ, the softening occurs no longer in patches, but presents those remarkable appearances described by M. Louis, as indicating the existence of pathological alterations. The forms of the softening in this case are those of stripes and bands of various dimensions occupying the situation of the plicæ. Wherever these stripes and bands exist, we find that the mucous membrane has been completely dissolved and the submucous coat laid bare. They have thus a blueish or silvery grey aspect, while the mucous membrane which they enclose may be of its natural colour, red, brown, or yellow, and appears in isolated patches of various forms and extent. It was the isolated and defined character of this form of softening which made it be considered as indisputably of a pathological nature. But the following explanation will show that it is a *post-mortem* lesion, and the consequence of the chemical action of the gastric acid. The mucous membrane possessing only in a very limited degree the power of diminishing its bulk, is always thrown into the form of plicæ when the muscular coat has contracted so as to diminish considerably the capacity of the stomach. When a quantity of gastric acid is collected on the surface of the mucous membrane in this state, it is obvious that the dissolvent property of this fluid will be exerted principally, if not exclusively, on the projecting borders of the plicæ, their lateral surfaces being protected from its action in consequence of their contiguity or the mucus collected between them. Hence it follows, that when the stomach is removed from the body, emptied of its contents and spread out, the plicæ are effaced, and the stripes and bands, not before observed, make their appearance. That this is the manner in which this form of softening is produced, is readily demonstrated by placing a quantity of gastric acid or digested food on the mucous membrane of a stomach in which the plicæ are well marked. In a few hours the appearances I have described will be conspicuous.

The presence of gas in the stomach gives rise to another form of softening which requires to be noticed. The softening terminates in a well-defined abrupt margin, beyond which the mucous membrane is found to present (so far, at least, as the gastric acid is concerned) its natural colour and consistence. The regular and defined margin of the softening is determined by the gas acting as a foreign body, equalizing the distribution of the acid, and confining its operation to a circumscribed portion of the mucous membrane.

Colour.—There are several important modifications of colour which accompany softening from the chemical action of the gastric acid. As they have already been described under the head of *Spurious Melanosis*, I shall only remark that they depend on changes taking place in the blood of the vascular system of the stomach, where this fluid is submitted to the action of the gastric acid. The modifications of colour thus produced are observed in the mucous membrane and bloodvessels. In the former the colour varies from dull yellow to orange, brown, or black, according to the quantity of blood present; in the latter, from light to deep brown or black. An opposite state, or extreme paleness

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of the softened mucous membrane, characterises that variety of *post-mortem* softening generally observed in infants and young children, and in emaciated, cachectic, and leucophlegmatic persons, in whom the blood is not only small in quantity, but contains a great disproportion of serum. In such individuals the whole stomach often appears as if macerated ; is, indeed, sometimes infiltrated with serosity, and is so completely deprived of blood that hardly any trace of this fluid is perceived, except in some of the large venous branches. These circumstances explain the occurrence of pale softening, or the ramollissement gélatiniform of M. Cruveilhier. Redness of the softened mucous membrane is never observed, its existence being incompatible with the chemical action of the gastric acid.

As the physical characters of the chemical dissolution of the coats of the intestines resemble those observed in the stomach, they do not require to be described. It is, however, important to remember that the softening occurs, in general, in the large intestines, and in that portion of them situated in the left hypochondriac region. From perforation of the intestine occurring in this situation,—from *without inwards*, and being accompanied with perforation of the stomach, in the cases which I have observed, I am strongly inclined to believe that it is generally the consequence of the gastric acid which had escaped from the latter organ.

From this description of the physical characters of the *post-mortem* effects of the gastric acid, and from the similarity which exists between these characters and those observed in healthy animals submitted to experiment, as well as in man when suddenly deprived of life by external injury, I think it must be admitted that a false interpretation has been given of the softening, erosion, and perforation of the digestive organs, by describing them as the effects of a peculiar disease. And when we reflect that all these lesions have been met with of every possible degree, extent, and form, in the bodies of those who have died of acute and chronic diseases, whether of one or of several organs ;—whether the stomach has manifested any signs of disease during life or not ;—in the total absence of all gastric symptoms, when one half of the stomach is destroyed, the liver, spleen, and intestines partly dissolved, and the diaphragm perforated ;—in the most robust as well as in the feeble and emaciated, and at every period of life ;—surely it would not be consistent with the principles of sound reason to ascribe them to any other than to a common agent, such as that which experiment has shown to be capable of producing them under all these adverse circumstances. Still the question has been urged—why do these lesions so frequently occur in children, in whom the principal, if not the only symptoms observed, are referable to derangement of the functions of the stomach and intestines ? But this question may be met by the following. How does it happen that the same lesions *do not occur* in cases in every respect the same as the former, as regards the derangement of function and the organs affected ? The answer is simply this—the presence in the former, and the absence in the latter case, of the chemical cause of these lesions. Gastritis or gastro-enteritis is the disease which exists in both cases, and would never have been described as anything else, but for the *post-mortem* effects of the gastric acid which I have described.

DESCRIPTION OF THE PLATES.

PLATE I.

Both figures in this Plate represent the *post-mortem* effects of the gastric acid on the coats of the stomach and on the blood, and illustrate the description of spurious melanosis given in the Fourth Fasciculus, and of that kind of softening accompanied with discoloration of the blood, described in the present Fasciculus. *Fig. 1* represents the stomach laid open in the direction of the small curvature; A, inferior portion of the œsophagus; B, commencement of the duodenum; C, fundus of the stomach. The different shades of colour which take place in the mucous membrane and in the blood were peculiarly well marked in this case, and are seen occupying the whole surface of the stomach, except towards the large curvature, where a small portion, E, of the mucous membrane, which had not been in contact with the gastric acid, has preserved its natural colour and consistence. This part contained gas. At the pyloric portion of the stomach, D, the mucous membrane presents the uniform dull yellow colour, or first change of colour produced on the blood by the gastric acid, and has not lost much of its natural consistence. At FFF, the change of colour deepens, and is accompanied by a pulpy state of the mucous membrane, both of which, from the form which they assume at the pyloric portion of the stomach, are seen occupying this membrane, where it had been thrown into folds or plicæ. At GG and HH, the black ramiform and punctiform discoloration of the blood are peculiarly well marked, and likewise at K in the vessels which ramify at the cardiac orifice and stretch into the inferior portion of the œsophagus. The softening is no where so conspicuous as at the fundus, where all the coats of the stomach, except the peritoneal, are completely dissolved, and presenting that gelatinous transparency so characteristic of this kind of softening. *Fig. 2* exhibits the same lesions, but occurring in a stomach affected with inflammation, and terminating in double perforation of the fundus. A, termination of œsophagus; B, commencement of the duodenum; C, fundus of the stomach. In this case the quantity of the gastric acid contained in the stomach was much smaller than in the former, and the softening as well as the discoloration is confined to the fundus and a portion of the body of this organ. The limits of the softening are marked by the irregular margin of the mucous membrane, within which all the other tunics are converted into a transparent yellowish-grey pulp, streaked with brown and traversed by a great number of the bloodvessels FFF, presenting the ramiform and punctiform black discoloration of the blood. D and

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E are the two perforations formed by the dissolution of the peritoneum. The inflammatory redness of the mucous membrane GGG, occupies the circumference of the softening, and extends to the pyloric portion H of the stomach, which presents its natural colour.

PLATE II.

Fig. 1 represents a variety of pale softening of the greater part of the mucous membrane, and two perforations of the fundus of the stomach. A, termination of the œsophagus; B, commencement of the duodenum; C, the two perforations, the fringed margins of which are formed externally by the peritoneum DD, internally by the muscular and submucous coats EE. The stripes or bands, FFF, formed by the dissolution of the mucous membrane of the plicæ, are particularly well seen in this figure, as well as the isolated portions of this membrane, GG, the blood in the capillary system of which has undergone the dull yellow or pale orange discoloration. The same discoloration is observed in the mucous membrane of the fundus. The brown and black ramiform and punctiform discoloration are also conspicuous in the submucous tissue generally, but particularly where this tissue, which is recognized by its grey silvery aspect, forms the bottom of the stripes alluded to above. It is worthy of notice that the blood in the vessels which occupy the inferior portion of the œsophagus, has preserved its red colour from the gastric acid not having reached it in this situation. *Fig. 2* is a representation of pale softening terminating in perforation of the stomach from a child. It is, however, often much paler than it is here represented. A, cardiac, B, pyloric orifice; C, perforation of the fundus; D, the fringed edges of the peritoneum; E, the muscular coat terminating in irregular prolongations; F, the mucous coat, the margin of which is also irregular. *Fig. 3* has been given as an example of softening from the chemical action of the gastric acid, such as it generally occurs in the stomach of the rabbit. The stomach is represented inverted; A, œsophagus; B, pyloric portion; C, body; D, fundus of this organ. The body and pyloric portion of the stomach were filled with undigested food, and in these situations the mucous membrane presented the colour and consistence natural to it in this animal during the act of digestion. The fundus, on the contrary, was filled with digested food and gastric acid, and in this situation all the membranes were more or less softened or entirely destroyed. The mucous and submucous coats had disappeared to an extent which was indicated by an irregular margin formed by the softened edges of both, as represented in the figure. The muscular coat, D, was thus entirely exposed, pale, pulpy, and transparent nearly throughout, and completely dissolved at the most depending part of the fundus C, where the peritoneum had also undergone the same degree of dissolution. *Fig. 4* is a representation of the internal surface of a portion of the colon, exhibiting the appearances which I have described as accompanying pale inflammatory softening of the mucous membrane and follicles of this intestine. AA, enlarged follicles; BB, circular patches formed by the destruction of the follicles, the mucous membrane being still entire; at CC this membrane is also extensively destroyed, and the grey submucous tissue laid bare around the whole circumference of the intestine, except at D, where small irregular portions of the mucous membrane remain.

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PLATE III.

This Plate represents red and pale inflammatory softening of the brain. *Fig. 1* red inflammatory softening of one of the hemispheres of the brain, from which a portion of the membranes has been removed, to show the change of form which takes place in the convolutions. AA, two of the convolutions soft and flattened; BB, other convolutions, greatly softened and quite sunk; portions of the cerebral substance are converted into a creamy-looking substance, which at C has been succeeded by hemorrhage; BB, the general increased vascularity of the membranes. *Fig. 2* is a longitudinal section of the same hemisphere, the appearances seen in the upper portion of which are strikingly illustrative of what might be called *acute inflammatory hemorrhagic softening*. A, anterior, B, posterior lobes; C, brown substance of one of the thalami; DD, brown substance of the convolutions; EE, white substance injected; F, summit of the hemisphere in which the extent of the softening is marked by the pale yellow colour of the white substance of the brain. The softening is greatest in the points where congestion and hemorrhage have taken place. The change of form and great breadth of the hemisphere in the direction of the softened part are very conspicuous. *Figs. 3 and 4* are examples of pale inflammatory softening. In *fig. 3* the softening affects the septum lucidum and the surface of the thalami in the third ventricle. A, internal lateral surface of the left hemisphere; B, cerebellum; C, medulla oblongata; D, cut surface of the left crus cerebri; E, optic nerves; F, G, pituitary and pineal glands, the latter much enlarged; HH, corpus callosum, cut longitudinally; K, right lateral ventricle and septum lucidum; L, pale inflammatory softening of the septum, a considerable portion of which is broken down into ragged patches of the consistence of thick cream or soft curd. At one point it is completely destroyed, and a communication is formed between both lateral ventricles. M, the same state of softening of the white substance on the surface of the optic thalami in the third ventricle. *Fig. 4*, a small portion of one of the hemispheres of the brain, showing that variety of pale softening of the brown substance of the convolutions which not unfrequently accompanies meningitis. A, some of the convolutions covered by the arachnoid and pia mater; B, other convolutions, from which these membranes, CC, have been removed; DD, solutions of continuity produced in the softened brown substance of the convolutions whilst separating the membranes, on the surface of which, at EE, are seen the portions of this substance which have been thus detached.

PLATE IV.

The figures in this Plate represent softening of the brain from obliteration of the arteries. *Fig. 1*, a portion of the right hemisphere; A, the lateral ventricle laid open; B, thalamus; C, corpus striatum of the right side. The upper half of this body was converted into a soft pulp of a pale straw and light brown colour, in which two small arteries, D, were seen ramifying. They were completely obliterated, and felt as firm to the touch as fine wires. *Fig. 2* represents the principal branches of the carotids, AA, in this case, in which the circulation was obstructed to a considerable extent, chiefly from fibrous substance contained within them. The situation of this substance is recog-

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nized by the pale spots seen on the external surface of the arteries. Two of these vessels are represented laid open at B and D, where the fibrous substance is seen attached to their lining membrane; C and E are transverse sections of the same vessels, nearly the whole caliber of which is seen to be occupied by this substance. *Fig. 3*, a section of part of the left hemisphere of the brain, in which the same kind of softening is seen, accompanied with orange-brown discoloration. The softening and discoloration are almost entirely confined to the brown substance of the corpus striatum, A, and that of several of the convolutions, BBB. In one of the latter and also in the corpus striatum, excavations are formed, in consequence of the complete destruction of this substance. The discolouration which accompanies the softening in this case as well as in the former is the result of sanguineous effusion, and the subsequent changes which take place in the blood which I have before noticed. C is the principal arterial trunk, some of the larger branches of which are obliterated, others obstructed by the presence of bony and fibrous formations; only two or three of the smallest branches are pervious. *Fig. 4*, softening of the pons Varolii; A, a transverse section of the pons; B, a longitudinal section of the medulla oblongata; C and DD softening of the brown and white substance. At C these substances were entirely destroyed, and an excavation formed sufficient to contain a cherry-stone; at DD they were in a pulpy state, and in both situations there was effused blood, which preserved its red colour, the sudden death of the patient not having afforded time for the changes of colour which are observed to take place in this fluid under opposite circumstances, as in the preceding case. The softening of the pons was the consequence of obstructed circulation in the basilar artery, as shown in *fig. 5*; A, the pons; B, the medulla oblongata; C, the basilar artery; D, left vertebral, E, right vertebral artery. This branch of the vertebral artery and the branch F of the basilar are much smaller than those of the opposite side, and were obliterated by a fibrous substance contained within them. At two points, G, of the basilar artery a similar substance existed, but did not entirely prevent the blood transmitted by the left vertebral from passing through it. The circulation however, was obstructed to such an extent as to occasion the cessation of nutrition, and softening of a portion of the pons. The morbid condition of the basilar and right vertebral arteries is represented in *fig. 6*, DDD. At B, one of the branches of the basilar artery is plugged up by a conical portion of fibrous tissue which was attached to a point C of this artery.

Plate 1.

Fig. 1.

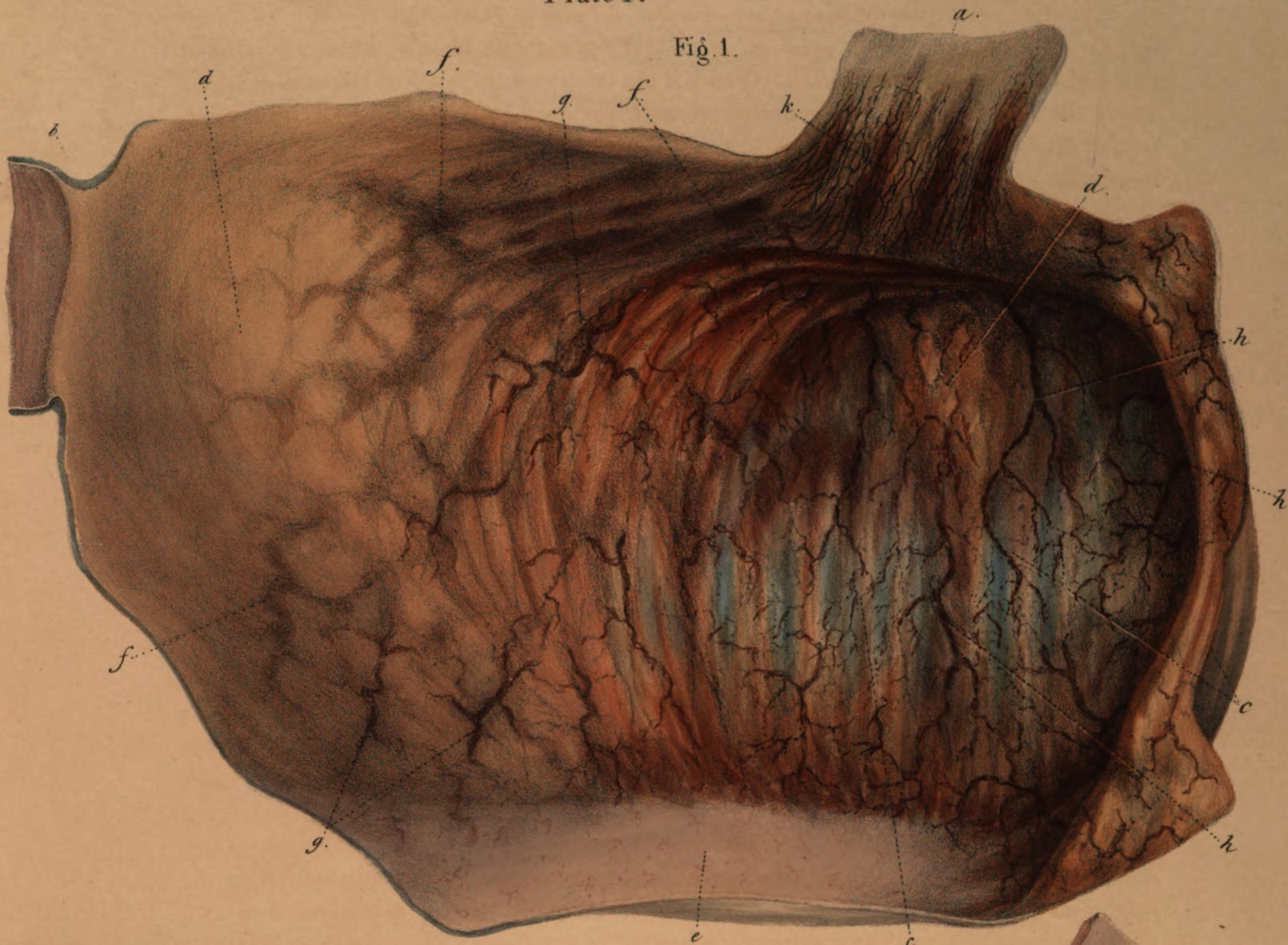
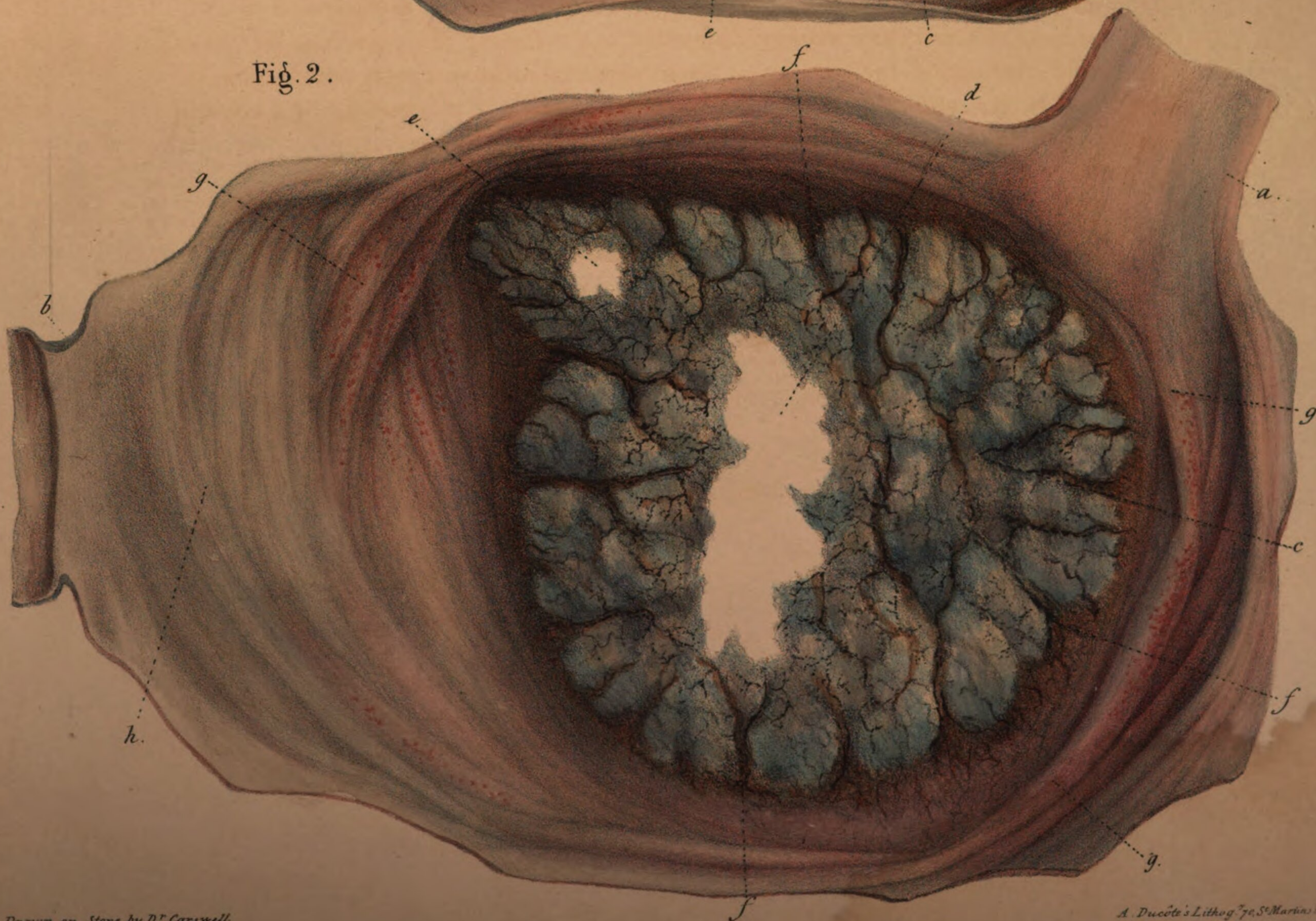


Fig. 2.



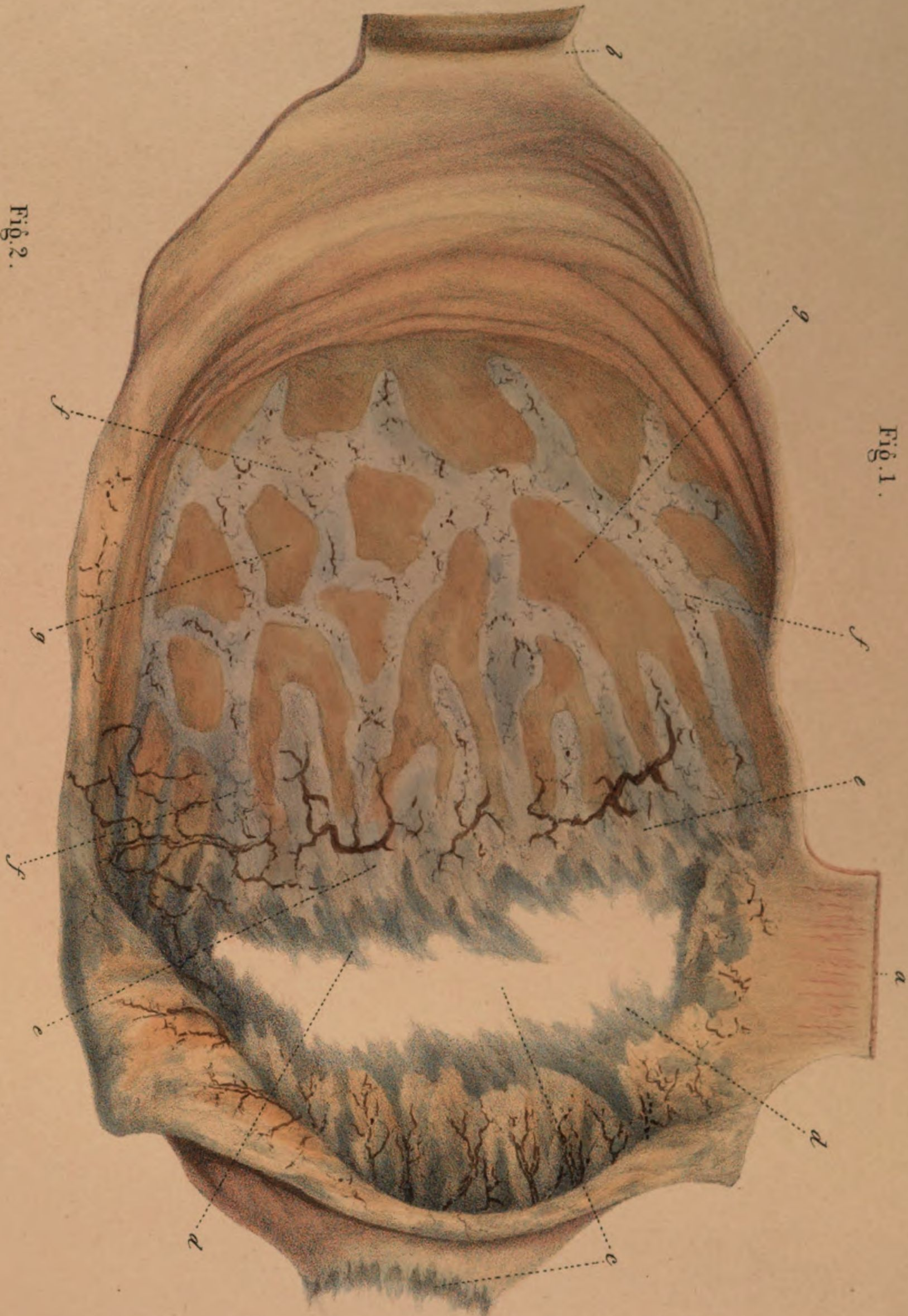
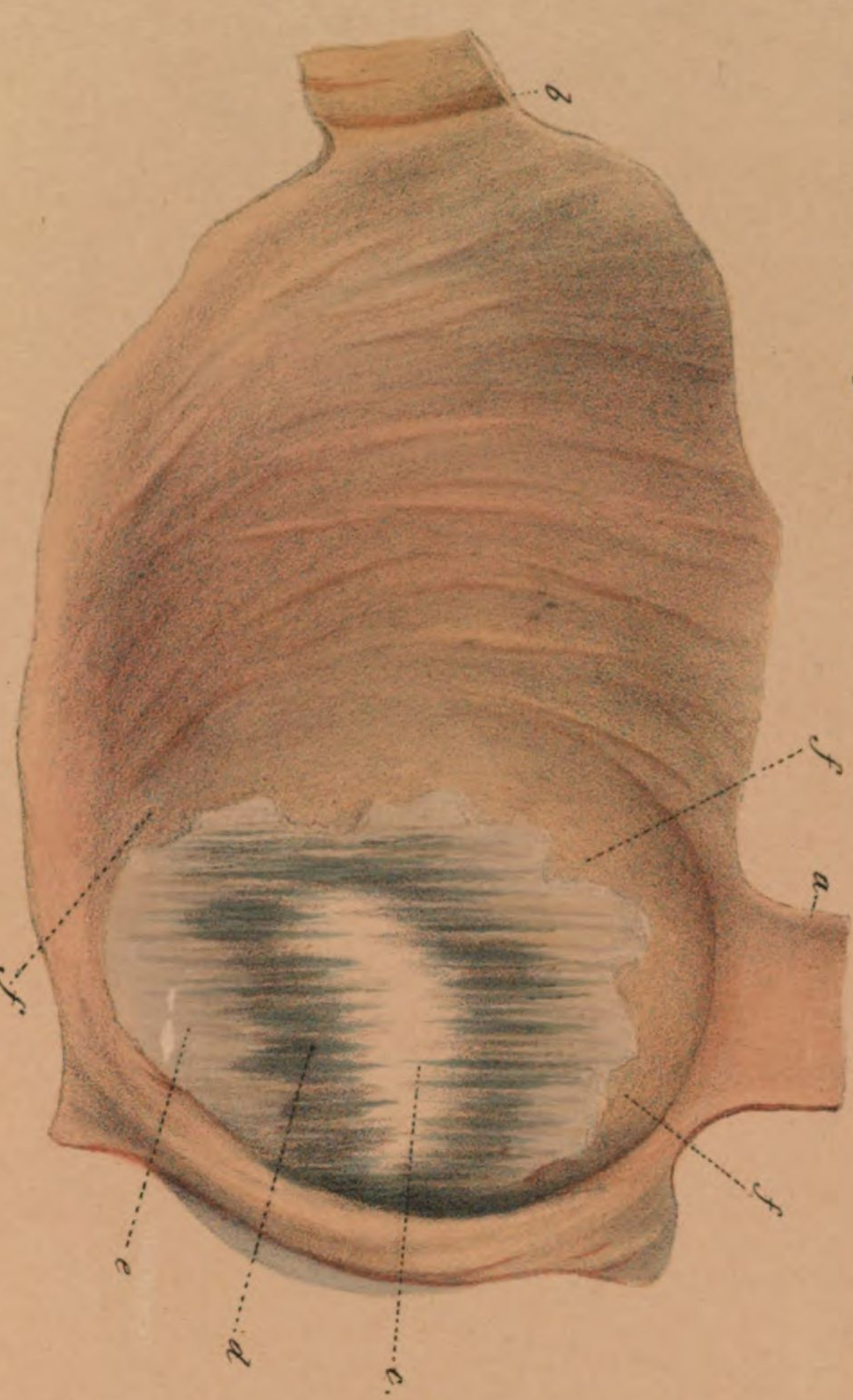


Plate III.



Fig. 3.

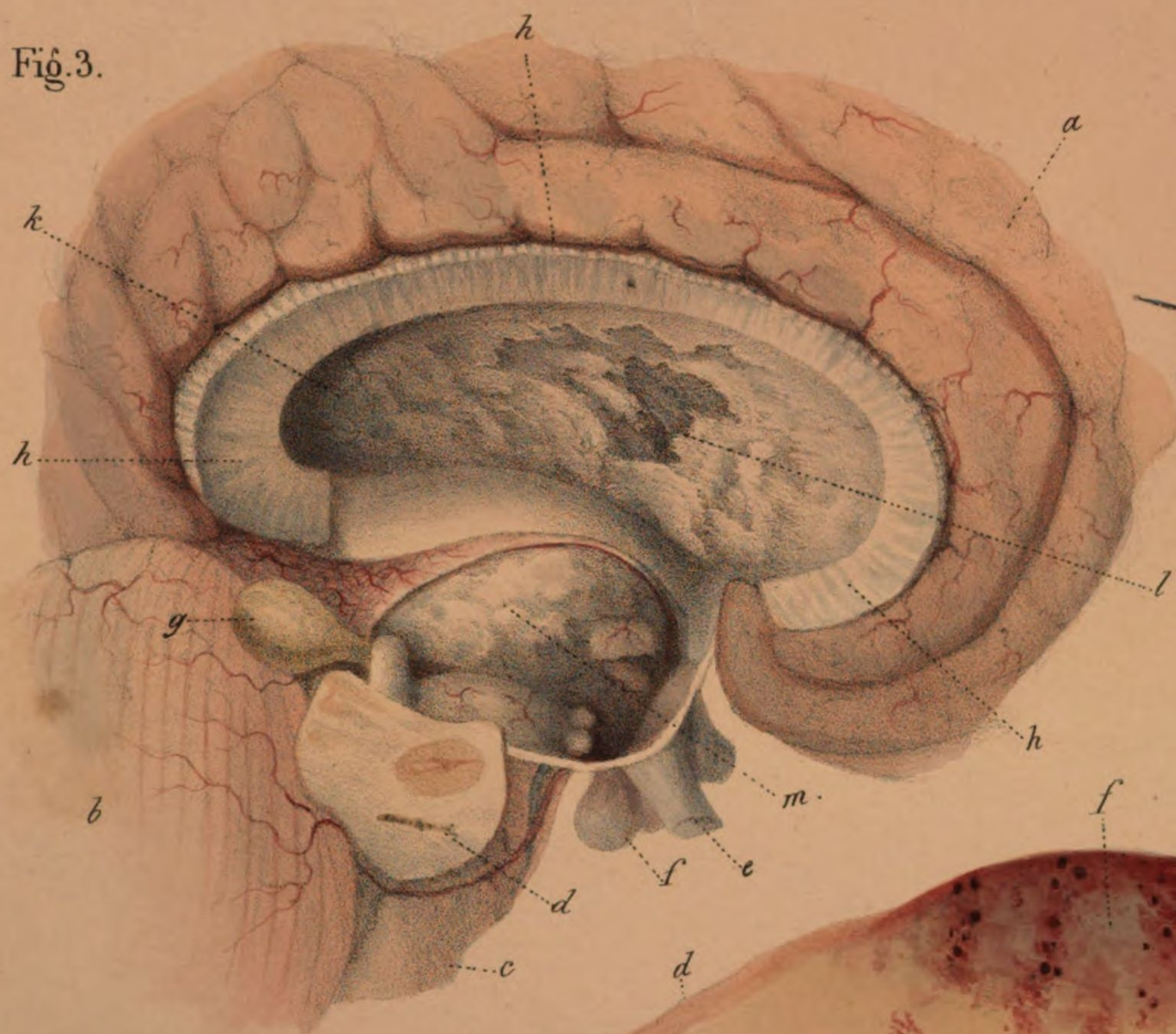


Fig. 4.

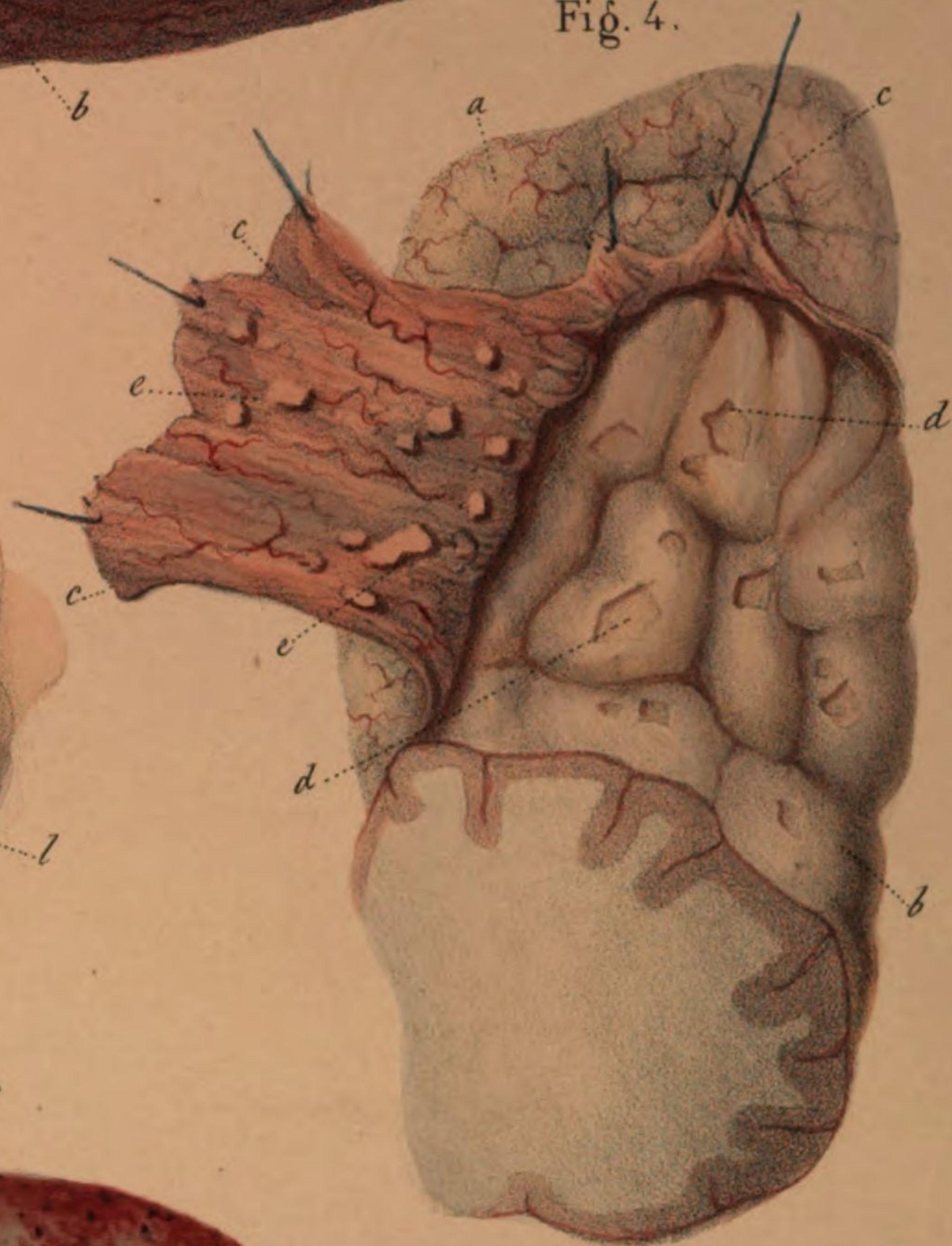


Fig. 2.



Plate 1V.

Fig. 3.



Fig. 1.

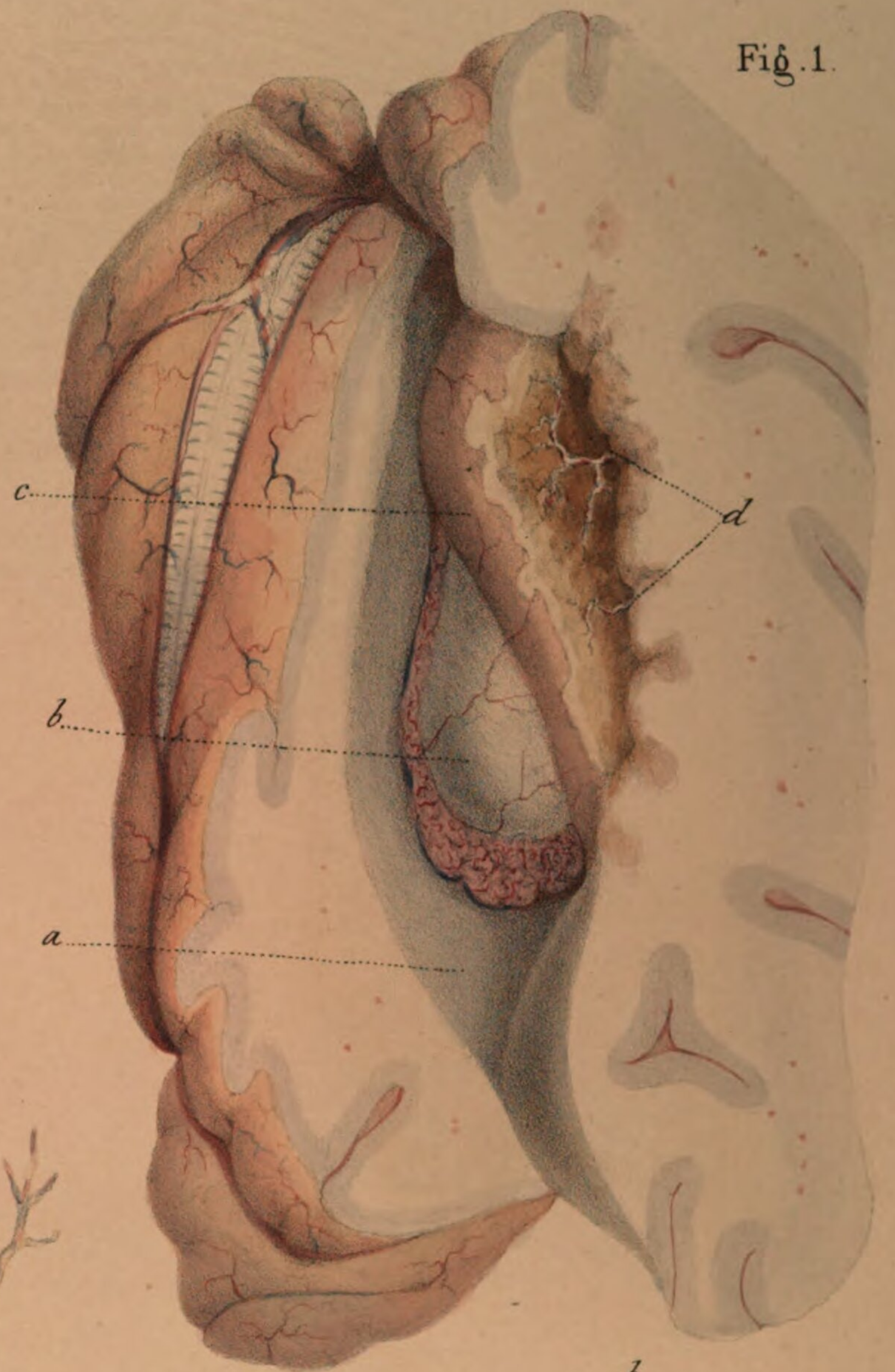


Fig. 2.



Fig. 6.

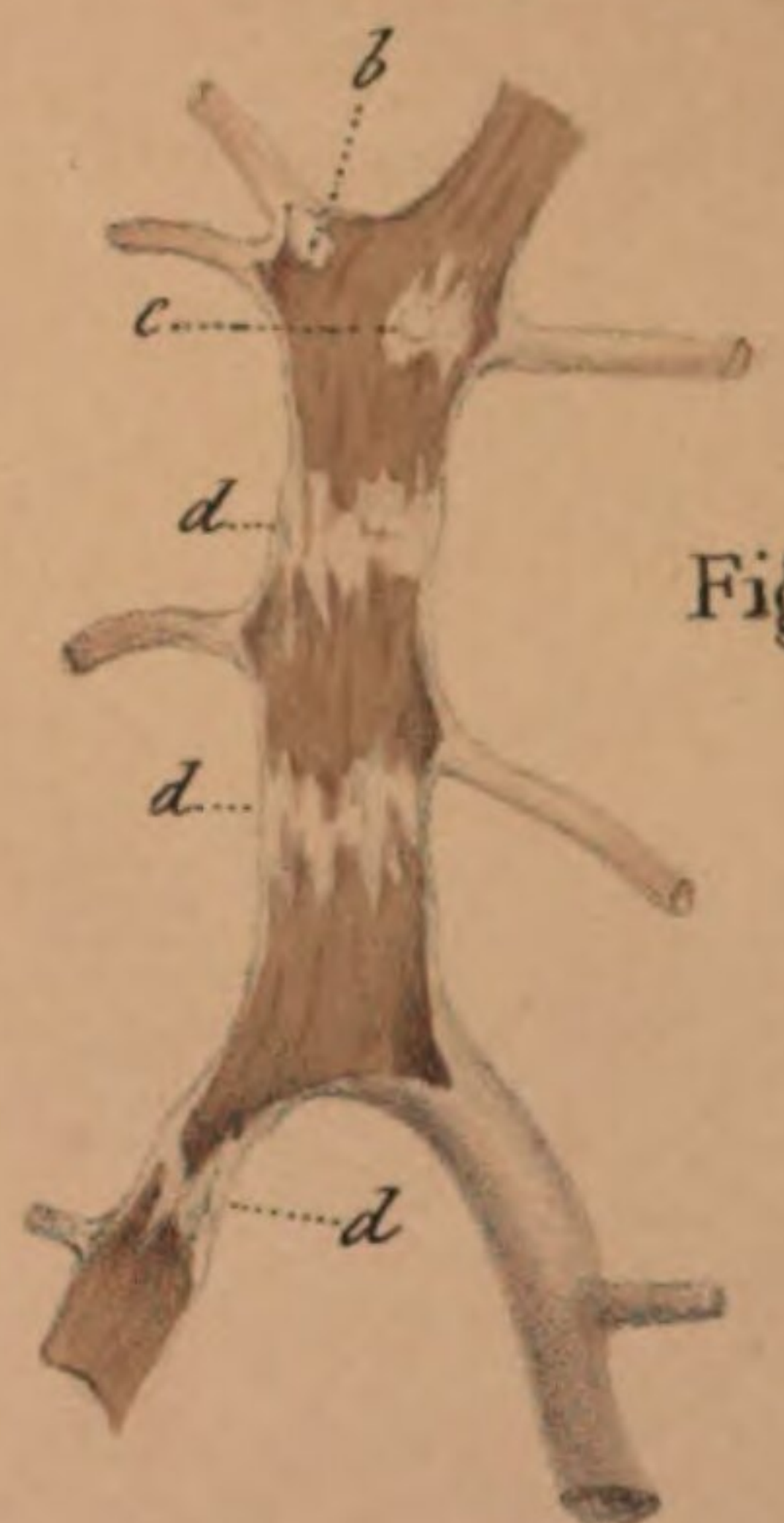


Fig. 4.

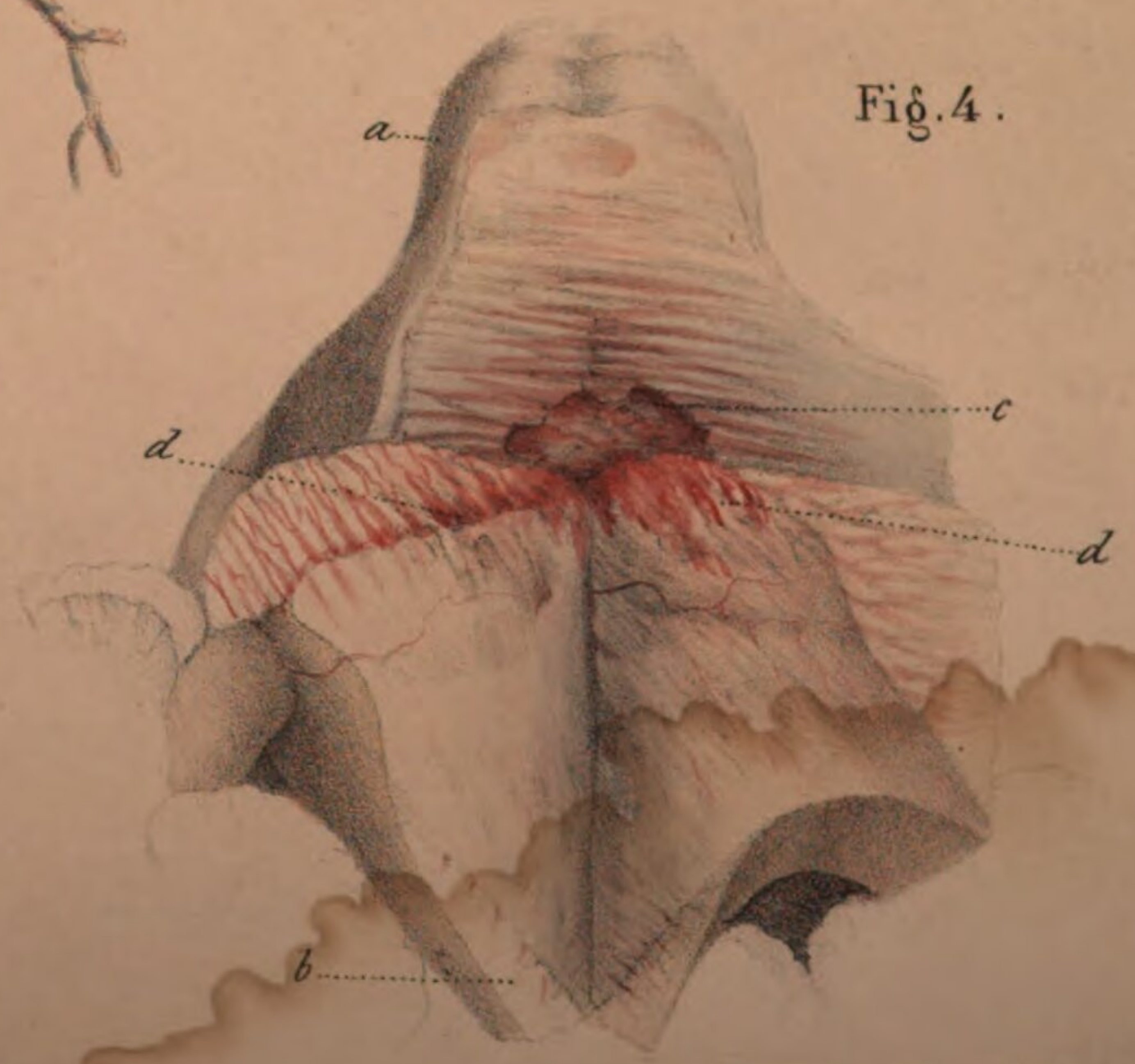
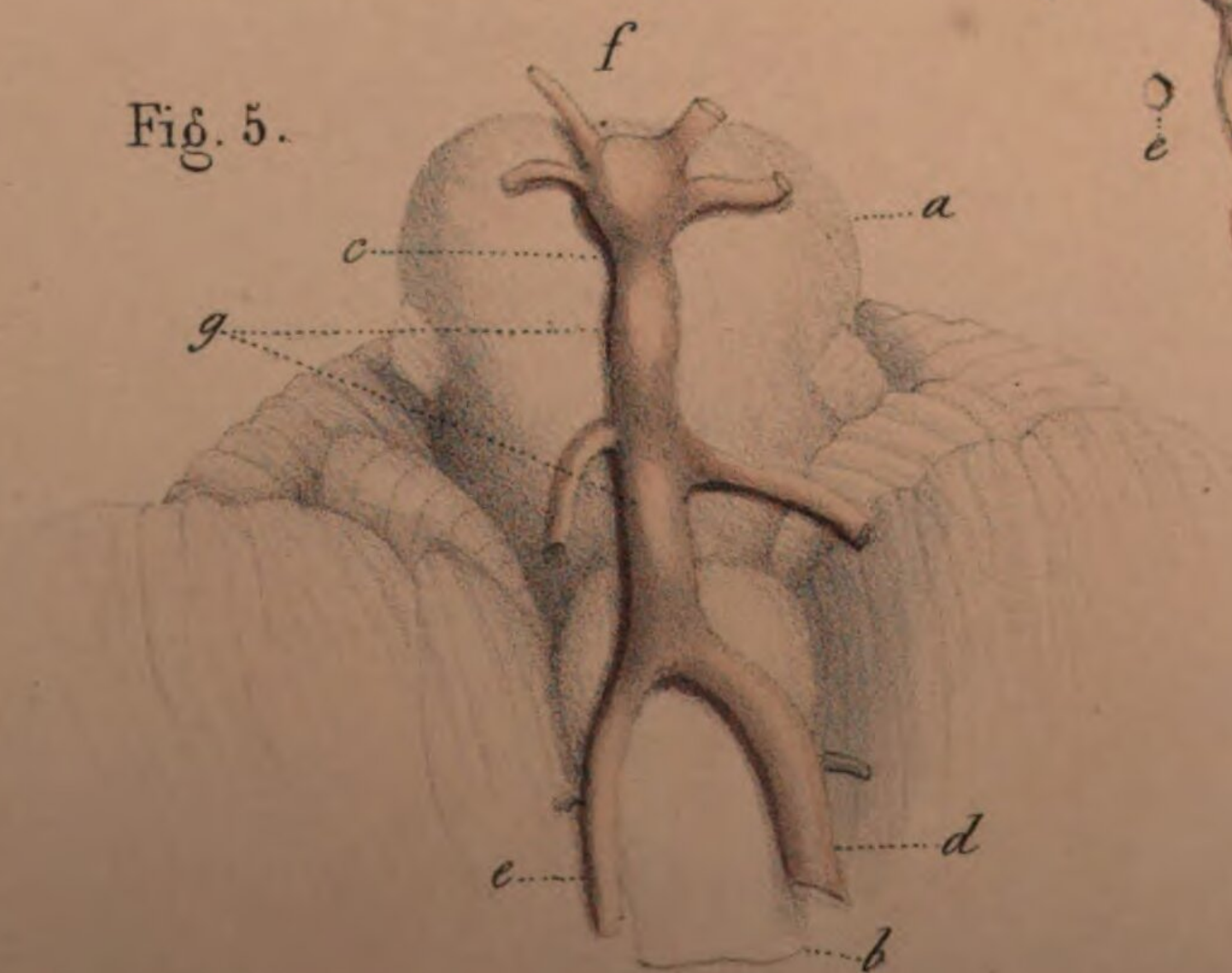


Fig. 5.



MELANOMA.

A MORBID product, presenting a black colour of various degrees of intensity, somewhat humid, opaque, possessing the consistence and homogeneous aspect of the tissue of the bronchial glands of the adult, was first described by Laennec under the appellation of *Melanosis*. Various and very different morbid products have since been described by several pathologists as examples of melanos, and even some *post-mortem* lesions have been confounded with this disease, in consequence of their presenting, as the most remarkable of their physical characters, a greater or less degree of blackness. To reject all melanotic formations which do not agree in all their characters with those assigned to melanos by Laennec, or to collect them indiscriminately into one entire group under the same appellation as has been done by others, would obviously render the pathology of these formations equally inaccurate and incomplete. While, therefore, I include under the title of Melanoma all melanotic formations, black discolourations or products, described by Laennec and other authors, I shall, however, in order to mark in a more especial manner the difference of their nature, separate them into two great groups, those of the first group being distinguished by the appellation of *True Melanos*, and those of the second group by that of *Spurious Melanos*. Thus, when these formations or products depend (as is the case with some of them) on a change taking place in that process of secretion whence the natural colour of certain parts of the body is derived,—or in other words, when they constitute what is called an idiopathic disease, I shall consider them as belonging to the first group; and when (as is the case with others) they originate in the accumulation of a carbonaceous substance introduced into the body from without, the action of chemical agents on the blood, or the stagnation of this fluid, I shall include them in the second group. There are several black discolourations which might also have been included in the present systematic arrangement, such as those observed in tissues affected with mortification, that have been subjected to the action of intense heat or powerful escharotics of various kinds; but as they never have been confounded with any of the forms of melanos, I shall not take any further notice of them in this place. We have, therefore, to describe—I. True Melanos, of which there is only one kind: II. Spurious Melanos, of which there are three kinds; 1st, from the introduction of carbonaceous matter; 2d, from the action of chemical agents on the blood; and, 3d, from the stagnation of the blood.

I. TRUE MELANOS.—*Definition of True Melanos*.—True melanos consists in the formation of a morbid product of secretion, of a deep brown or black colour of various degrees of intensity, unorganized, the form and consistence of which present considerable variety solely in consequence of the influence of external agents.

Seat, Mode of Formation, and Origin of True Melanos.—The most frequent seat of true melanos is the serous tissue, more especially where this tissue constitutes the cellular element of organs. Here the melanotic matter is formed after the manner of secretion, accumulates in the cells of which the serous tissue is composed, and gradually acquires the form of tumours of various sizes. A similar mode of formation of this matter is observed to take place much more conspicuously in loose cellular tissue, and particularly on large serous surfaces, such as those of the pleuræ and peritoneum.

[*Melanoma*, 1.]

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The next variety observed in the seat and mode of formation of the melanotic matter, is that in which this matter is deposited into the substance or molecular structure of organs, after the manner of nutrition. And, lastly, the melanotic matter is found in the blood contained chiefly in the venous capillaries, and under circumstances which show that it must have been formed in these vessels. The much greater frequency of melanosis in the grey and white than in the bay, brown, or black horse, is a circumstance which may be noticed here as favourable to the theory which ascribes the origin of this disease to the accumulation in the blood of the carbon which is naturally employed to colour different parts of the body, as the hair, rete mucosum, choroid, and other parts. This theory is supported by the results of the chemical analysis of melanosis, as well as by the facts derived from the consideration of the physical and anatomical characters of the disease, both of which also concur in confirming the opinion I have expressed regarding its seat and mode of formation.

Physical Characters of True Melanosis.—The physical characters of this disease comprehend the form, bulk, colour, and consistence which it presents in different organs.

Form.—True melanosis occurs under four varieties of form, which I have denominated as follows:—1st, Punctiform; 2d, Tuberiform; 3d, Stratiform; 4th, Liquiform.

1. *Punctiform Melanosis.*—This form of the disease appears in minute points or dots, grouped together in a small space, or scattered irregularly over a considerable extent of surface. These appearances are most frequently seen in the liver affected with melanosis, the cut surface of this organ appearing as if it had been dusted over with soot or charcoal powder. When examined by the aid of a lens, the black points sometimes present a stellated or penicillated arrangement, which, in some cases, can be distinctly seen to originate in the ramiform expansion of a minute vein filled with the melanotic matter. At other times this matter appears to be deposited in the molecular structure of this organ in a manner similar to that of the organizable part of the blood. In such cases it consists of the most minute points disseminated throughout the acini of the liver, which then assume a uniform grey aspect of various depths of shade, terminating in black.

2. *Tuberiform Melanosis.*—This is by far the most common of all the forms of melanosis; it occurs in most of the organs of the body, and also sometimes on serous surfaces, as the pleura and peritoneum. In the former situation the tumours are generally globular, and in the latter are not unfrequently pyriform. They are most frequently found single in organs and aggregated in cellular and adipose tissues, and have, perhaps, never been found limited to one organ, the deposition of the melanotic matter taking place simultaneously or successively in a great many organs, or in the cellular tissue of the different regions of the body. The melanotic tumours are most numerous in the cellular and adipose tissues, and from their aggregation produce lobulated or irregularly shaped masses of great bulk. The tuberiform melanosis is always combined with the punctiform in the liver, lungs, and kidneys, and sometimes appears to have the same mode of origin as the latter; that is to say, in the deposition of the melanotic matter after the manner of nutrition. On serous membranes the melanotic tumours are, on the contrary, accompanied by the liquiform variety of the disease yet to be described, and may, like it, be seen to originate in a process of secretion, or in the deposition of the black matter on the free surface of these membranes.

The melanotic tumour is described as encysted when enclosed by a membranous covering, and non-encysted when it lies in immediate contact with the tissue of the organ in which it is contained. It is, perhaps, never found encysted in compound tissues or organs, as the brain, lungs, liver, and kidneys; whereas it is always so in the cellular

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and adipose tissues, and, as we have seen, sometimes also on the surface of serous membranes. In the former situation the cyst is formed by the gradual distension and condensation of the cellular tissue in which the melanotic matter was originally deposited; in the latter, it may be formed either by a prolongation of the serous membrane, (when the melanotic matter is deposited, for example, under the pleura,) or of cellular tissue of new formation. The latter origin of the cyst is, perhaps, the most common on the free surface of serous membranes, the cellular tissue of which it is composed being formed during the deposition of the melanotic matter.

3. *Stratiform Melanosis*.—This form of the melanotic deposit occurs only on free surfaces: it presents two degrees or stages. In the first, the black matter is so sparing in quantity, that the serous membrane on which it is deposited presents an appearance of having been painted or stained with a deep brown or black colour. In the second, the black deposit is more abundant, and forms a distinct layer on the surface of the serous membrane above which it slightly projects. The consistence of the matter thus deposited resembles in general that of jelly, and is enclosed, as I have already said when speaking of the preceding form of melanosis, either in a soft spongy cellular tissue, or fine transparent serous membrane of new formation; so that, when pressed, it feels pulpy, but is not removed by the finger or a scalpel passed over it, unless some force is employed.

This form of melanosis is described by Andral and other pathologists as occurring frequently on the peritoneum in chronic inflammation of this membrane. Such, however, is not the case. The "*mélanoses déposés sous forme de couches solides à la surface des membranes*" of this author is of an entirely different nature from that which I am now describing under the appellation of the *stratiform*. It is the result of the chemical action of certain fluids and gases formed in general in the digestive organs, on the blood contained in the vessels of pseudo-membranes of the peritoneum, or effused into the substance of the one or on the surface of the other. The stratiform melanosis is rare in its occurrence and limited in its extent in man. In the horse, however, it is sometimes considerable in degree and extent, and is chiefly found on the peritoneum, pleura, and pericardium.

4. *Liquiform Melanosis*.—The appearance of true melanosis in a liquid form has, in general, been confined to natural or accidental serous cavities. Among the former, the cavities of the pleura and peritoneum furnish almost the only examples in which the liquid melanotic matter has been observed, and that too in very small quantity. I have never seen it in man as a product of secretion, but have met with it in consequence of the destruction of melanotic tumours and the effusion of their contents into serous cavities, the walls of which they had perforated. The accidental serous cavities in which it has been found are those which constitute cysts, particularly in the ovaries. Brechet, Andral, and Cruveilhier describe the occurrence of the liquiform melanosis on the surface of the mucous membrane or in the cavity of the stomach, but in doing so they have confounded it with the black discolouration of the blood produced by the action of the gastric juice on this fluid when effused.

Bulk.—The quantity of the melanotic matter deposited is often very considerable. The irregular or lobulated masses which it forms in the cellular tissue are sometimes larger than the fist, and the globular tumours to which it gives rise in parenchymatous organs may acquire the size of a small orange. The largest masses are found in loose cellular tissue, as the retro-peritoneal, and are always composed of a number of smaller ones; but single tumours of the largest size have been observed in the liver. Masses of true melanosis have been found in the former situation in the horse, to weigh from twenty to thirty, and even forty pounds.

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Colour.—The colour of melanosis, whether in the solid or fluid state, although always tending to black, frequently presents various shades of brown, such as that of chocolate, bistre, or China ink, having either the dull aspect of soot, or the glossiness of pitch. The deep black colour and glossy aspect are more frequently met with in inferior animals than in man, and in both, these appearances are most marked when the melanotic deposit exists in the form of a firm tumour. The quantity of cellular tissue intermixed with the melanotic matter, as well as the presence of a certain quantity of blood, give rise to a greater variety in the shade and depth of colour than this substance would otherwise present. Like all colouring matters in a fluid state, that of melanosis, when placed in contact with a white surface, communicates to it its peculiar tint, but the stain thus produced is readily removed by ablution.

Consistence.—The consistence of melanosis presents great variety. There are two circumstances which seem to determine the degree of consistence which this morbid product presents when examined *in situ*: these are the *texture* and *form* of the part in which it is deposited. Thus, it is never found solid in serous cavities, for the plain reason that its diffusion is not limited by dense unyielding tissues. Even in tumours attached to the serous covering of these cavities, it is, for the same reason, either perfectly fluid, or not more dense than animal jelly. Loose cellular tissue is also occasionally filled with the black matter in a fluid state. In the dense texture of the cutis, on the contrary, even the smallest tumours may be nearly as hard as cartilage, and are generally as firm as the pancreas. In the lymphatic glands and in the brain the melanotic tumour acquires only a medium degree of consistence, although it is generally firmer in the former than in the latter, in consequence of the capsule of the glands acting as a compressing cause. In the liver and lungs we perceive the same relation between the texture of these organs and the consistence of the melanotic tumours formed in them. In both they are pretty firm; much about the consistence of a lymphatic gland, from the black matter being contained either in the capillaries, molecular structure, or cellular texture of these organs.

From these facts it is obvious that the consistence of melanosis is entirely dependent on the nature of the part in which the black deposit is retained. That it is not, in itself, and from the moment of its deposition, a solid product as described by Laennec, is certain from what I have said of its formation on the free surface of serous membranes alone, on which there can be no doubt of its being deposited in a fluid state, and afterwards acquiring a greater or less degree of consistence from the presence of the accidental cellular tissue in which it becomes enveloped.

At an indefinite period of its formation, the solid melanotic tumour is, however, observed to lose its consistence, and to become ultimately converted into a soft or almost fluid mass. This change of consistence constitutes the softening process of Laennec, but which is not, as he believed it to be, of a vital nature and originating in the melanotic matter itself. It is effected by the destruction of the tissues which surround, or are enclosed within, the melanotic tumour, and the simultaneous effusion of serosity. So soon as these tissues are destroyed, the melanotic matter is deprived of its connecting medium or envelope, is gradually detached by the serosity to the effusion of which its presence, as a foreign body, gives rise, and is converted into a soft pulp or a dark brown or black fluid.

Inflammation rarely accompanies the softening process, and when ulceration and sloughing occur, they appear to be chiefly owing to the melanotic matter compressing or obliterating the bloodvessels of the tissues in which it is contained.

The following may also be enumerated as physical characters of the melanotic matter. This matter is quite opaque, and has no marked odour or taste. In its natural

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state, or when mixed with water, exposed to the air it becomes dry, brittle, and pulverizable, and does not emit the odour of putrefaction until after a long period. When burnt, it swells, gives out a great deal of smoke, a marked empyreumatic odour, and is converted into a carbonaceous substance.

Chemical Characters of True Melanosis.—The most complete analyses of the melanotic deposit that have been published, are those of Lassaigne, Foy, and Barruel in France, and Dr. Henry of Manchester. The two former chemists procured it from the horse, the two latter from man. According to Barruel, melanosis of the human subject is essentially composed of the colouring matter of the blood, united with fibrine, both of them “*se trouvant dans un état particulier*,” three distinct kinds of fatty matter, and a considerable quantity of the phosphate of lime and iron. Foy found a portion of a melanotic tumour from the horse composed of—

Albumen	15,00	Sub-phosphate of lime	8,75
Fibrine	6,25	Muriate of potash	5,00
A highly carbonized } principle, probably } altered cruor . . . }	31,40	Ditto soda	3,75
Water	18,75	Carbonate of soda	2,50
Oxyde of iron	1,75	Ditto lime	3,75
		Ditto magnesia	1,75
		Tartrate of potash	1,75

From the results of these analyses, which do not differ materially from others that have been obtained, it is sufficiently obvious that the melanotic matter is essentially composed of the constituent elements of the blood. Indeed, this conclusion is generally adopted; and it would appear from the analysis of Foy, that the colouring material of melanosis, *that* which gives to this morbid product its peculiar character, is a highly carbonized principle, bearing a considerable analogy to the colouring matter of the blood.

Anatomical Characters of True Melanosis.—It has already been shown in the description which has been given of the physical characters of melanosis, that the solid form which this morbid product assumes is owing to the presence of the tissues in which it is contained. We have seen that the melanotic matter in the form of a round tumour may lie in immediate contact with the substance of the organ in which it is deposited, or may be bounded by a membranous capsule. When a single tumour of the latter description is divided, and a quantity of the melanotic matter is removed from it by pressure and ablution, a multitude of fine filaments and lamellæ are seen connected with the capsule, traversing its contents in every direction, and presenting an appearance resembling cellular tissue when distended with serosity.

When a number of melanotic tumours are grouped together, they are included in a common capsule, and separated from one another by their respective coverings, and portions of cellular tissue contained in the angular spaces sometimes left between them. It is in these filamentous and cellular tissues alone that bloodvessels or nerves are to be seen. Minute arteries and veins may be observed ramifying in both, but they never pass beyond the limits of these tissues. Large branches, and even trunks of arteries and veins, are sometimes found passing over the surface, or included in the aggregated masses of melanotic tumours.

There is a great difference in the relative quantity of cellular tissue and melanotic matter which constitute these tumours. The cellular tissue is generally small in quantity, in some cases hardly to be perceived, and in others, of rare occurrence, much more abundant than the melanotic matter which it contains. However, when this last case occurs, it is generally owing to the deposition of the melanotic matter in an accidental

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tissue, of which we have occasionally striking examples in fibrous, carcinomatous, and erectile tissues, having thus produced compound tumours, the structure and composition of which vary with the relative proportions of their respective materials, and of the melanotic deposit which they contain. Melanosis is more frequently combined with carcinoma than with any other disease, but there is no similarity of nature in these two diseases, their anatomical, physical, and chemical characters being totally different; several varieties of the former are highly organized, while the latter is an unorganized substance, injurious only from its quantity, the number of organs which it affects, its situation and mechanical operation. Examples of melanosis combined with fibrous, carcinomatous, and erectile tissues, are given in Plate I. *fig.* 4; Plate II. *figs.* 1, 2, 4 and 5; and Plate III. *fig.* 1.

II. SPURIOUS MELANOSIS.—A. *From the Introduction of Carbonaceous Matter.*—This kind of spurious melanosis occurs only in the lungs.

Physical Characters.—Both lungs present one uniform black carbonaceous colour, affecting nearly all the tissues of these organs. The bronchial glands partake also of the same black colour. The pulmonary tissue is more or less indurated and friable, infiltrated with black serosity, and broken down in several parts into irregular excavations, sometimes of considerable size.

Chemical Characters.—The following are the results of a minute analysis, made by Dr. Christison, of a quantity of the black matter taken from a patient who died of this disease in the Infirmary of Edinburgh, under the care of the late Dr. J. C. Gregory, by whom the history of the case was published in the 109th No. of the *Edin. Med. and Surgical Journal*.

1. Concentrated nitric acid boiled on the black matter did not alter its colour.
2. Immersion in a strong solution of chlorine had also no effect.
3. A strong solution of caustic potass boiled on it took up some animal matter, and filtrated very slowly; the first part that passed through was opaque and black; but the last portions were of a pale yellowish-brown colour, and transparent; so that none of the black matter was dissolved. The black matter remained on the filter, and this, well washed and dried, burned like charcoal powder, without swelling up, and with scarcely any animal empyreuma, leaving a considerable pale-grey ash.
4. A small portion of black powder left after the action of boiling nitric acid was well washed, dried, and introduced into a minute glass ball, with a tube attached, which was subsequently drawn out by means of the spirit-lamp flame to a fine bore. On the application of a low red heat to the ball, there was disengaged, at the open end of the tube, a considerable quantity of gas, which had the odour of coal-gas, and on the approach of a light took fire and burned with a dense white flame. In the tube a dark yellow fluid likewise condensed, which had very exactly the odour of impure coal-tar naphtha, and became a soft mass on cooling, of the consistence of lard. This, when compressed between layers of filtering paper, yielded an oily stain to the paper, and left a white matter, which dissolved in boiling alcohol, and separated again, on cooling, in the form of minute obscure crystals. “In the product of this experiment,” says Dr. Christison, “it is scarcely possible not to recognize the ordinary products of the distillation of coal. A gas of the same quality was procured, and likewise a naphthous fluid, holding in solution a chrySTALLINE principle, analagous to, if not identical with, naphthaline.”

Although certain forms of black discolouration of the pulmonary tissue were supposed by Laennec to originate in the inhalation of the carbonaceous product of ordinary combustion,—an opinion previously entertained by Mr. Pearson,—there was no recorded fact that could be regarded as furnishing undeniable evidence of the accuracy of this

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opinion till the publication of this interesting case. The physical characters of this form of spurious melanosis,—viz. the uniform black colour of both lungs; the absence of any similar discolouration of any other organ; the occurrence of the disease in those habitually exposed to the inhalation of the coal-dust always contained in the atmosphere of a mine; and the black matter found in the lungs consisting essentially of this substance;—are circumstances which demonstrate clearly the origin of the black matter, and its identity with the carbonaceous powder inhaled with the air in breathing.

B. *From the Action of Chemical Agents on the Blood.*—The black discolouration of the blood which belongs to this division of spurious melanosis, is produced by the operation of an *acid* chemical agent. It is, consequently, met with only in those organs in which this agent exists as a healthy or morbid product, or to which its influence occasionally extends. Hence, as the stomach is the only organ in which an acid fluid is formed, and is, at the same time, a healthy product, this kind of black discolouration of the blood is no where so frequently observed, so conspicuous, and so extensive as in this organ.

The same discolouration of the blood occurs also in the intestines from the anormal formation of a fluid and gaseous acid product. It is owing to the proximity of the peritoneum to these normal and anormal acid products, that blood situated beneath this membrane, on its free surface, or in its cavity, undergoes so often the change of colour in question; and it is owing to the same circumstances of situation, that portions of the liver and spleen are so frequently found to present the same black colour.

The black discolouration of the blood may be effected during life or after death; but before it can take place during life, the blood must have ceased to circulate. This fluid may be contained in its proper vessels, or effused into the cavity of the stomach, intestines, or peritoneum. Under these circumstances the following are the physical characters by means of which the changes which it undergoes are recognized.

Colour.—The colour of the blood which has been submitted to the action of the acid contents of the digestive organs, varies from a dull yellow or orange tint to the colour of chocolate, bistre, or soot. Any of these colours may exist alone, or they may all be present at the same time. The brown and black colours are the most frequent in their occurrence, and are met with in all the situations which I have enumerated; but the yellow and orange are seldom seen except in the mucous membrane of the stomach.

Forms.—The forms which this kind of spurious melanosis presents are determined by those of the organs in which the blood is contained: they are the *Punctiform*, *Ramiform*, *Stratiform*, and *Liquiform*.

The *punctiform* and *ramiform* black discolourations of the blood have their seat in the capillaries and veins, and are best seen on the internal surface of the stomach, where they most frequently occur. They always occupy that part of the stomach in which the gastric acid is contained after death. The punctiform and ramiform discolourations of the blood present an appearance similar to what would be produced by injecting the capillaries and veins of a portion of the stomach with a mixture of chocolate or soot and water. The bloodvessels, trunks, branches, and capillaries, are sometimes completely filled; at other times only partially so, being continuous or interrupted, and thus forming lines or rows of brown or black dots, or minute points, the dots having their seat in the trunks or larger branches, and the minute points in the smaller bloodvessels or capillaries. These appearances may be limited to a small portion of the stomach, generally a portion of the fundus, or they may occupy the whole surface of this organ. Their extent is in proportion to the quantity of the gastric acid, and the degree of discolouration to the quantity of blood contained in the vascular

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system of the stomach. The black discolouration of the blood in the vessels of the stomach is always accompanied with the chemical dissolution of the coats of this organ. Hence the bloodvessels are generally laid bare by the destruction of the mucous and submucous coats of the stomach; and not unfrequently the coats of the vessels themselves are dissolved, and the blood left in the form of black points, dots, or striæ.

The ramiform black discolouration of the blood from the presence of an acid is seldom met with in the intestines, but the punctiform is not uncommon. The latter takes place in the capillaries of the villosities, and in those around the orifices and basis of the follicles. When the villosities are the seat of the discolouration, the surface of the mucous membrane appears as if it had been dusted all over with fine powdered charcoal, which gives to it a deep grey or slate colour. The discolouration may occupy the orifice of a number of contiguous isolated follicles, or of the aggregated follicles, and give rise to an appearance resembling acne punctata; or it may surround the orifice or basis of the isolated follicles, when it has an annular form.

These appearances observed in the villous and follicular structures of the intestine have always been confounded with those produced by chronic inflammation. Both lesions are so similar in their physical characters, that it is only from the absence or presence of an acid in the affected portion of the intestine that we can form a decided opinion on the nature of either.

The ramiform black discolouration of the blood is met with occasionally on the peritoneum, in cases of chronic tubercular peritonitis. The tubercles scattered over the surface of the peritoneum are surrounded by a dark ring, or a multitude of minute vessels, filled with black blood, either grouped close together, or having a stellated arrangement. The tubercles, if small, are thereby greatly obscured, and the peritoneum appears as if spotted with a deep brown or black pigment.

The *stratiform* black discolouration is most frequently observed on the peritoneum, in its sub-cellular tissue, or in false membranes, when blood has been effused upon the former or into the latter. The discolouration may occupy the greater part of the parietal portion of the peritoneum, or it may be confined to a small extent of that which covers the surface of the viscera. Sometimes the discoloured surface is as black as ink; at other times it presents a mixture of brown and black; and lastly, the effused blood may be occasionally seen of its red colour, and gradually passing into the brown or black.

The *liquiform* black discolouration of the blood is produced by the effusion of this fluid into the cavity of the stomach, intestines, and peritoneum. The colour of the effused blood in the stomach and intestines resembles that of chocolate, bistre, China-ink, or soot; it is sometimes of a watery, at others of a creamy consistence, or thick and ropy like tar, and varies in quantity from a few ounces to several pints. This condition of the blood is observed most frequently at the termination of carcinoma of the stomach, when softening, ulceration, and sloughing have taken place, followed by hemorrhage, and the blood is submitted to the chemical action of the gastric acid. Hence the *black vomit* which so often accompanies the last stage of carcinoma of the stomach. Whatever gives rise to hemorrhage in this organ, as follicular ulceration, sanguineous exhalation, or perforation of a bloodvessel, is accompanied by the same change in the colour of the blood, if the stomach contains a quantity of acid sufficient to produce this change, at the time the hemorrhage takes place. The same may be said of the black dejections which constitute melæna. In some cases of this kind I have found the large intestines deeply ulcerated, filled with a pitchy-looking substance, and the surface of the ulcers themselves, whence the hemorrhage proceeded, impregnated

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with a similar matter. The black discolouration of the blood in the large intestines and in the cavity of the peritoneum, depends, perhaps, more frequently on the presence of sulphurated hydrogen gas than an acid fluid, owing, no doubt, to this gas possessing the properties of an acid. The change of colour which the blood undergoes when collected in the cavity of the peritoneum is, however, nearly the same as that which takes place in this fluid when effused into the cavity of the stomach. The external portion of the blood, or that which is in contact with the peritoneal surface of the intestines, is always deepest in colour, being sometimes as black as pitch, whilst that which is more remotely situated is either of its natural colour or but slightly darkened.

C. *From the Stagnation of the Blood.*—Black discolouration of the blood has been long known to follow as a consequence of retarded or interrupted circulation. This change in the colour of the blood is never so conspicuous as when it takes place in the extreme venous circulation or in the capillaries. When the blood has ceased to circulate in the latter vessels, it coagulates, and the serum is forced out along with the salts, which are absorbed. That which remains is an almost black substance, of the consistence of firm fibrine, and is probably composed in great part of this animal principle and hæmotosine. The production of the black colour would seem to be the consequence of the removal of the salts, from which, as has been clearly demonstrated by Dr. Stevens, the red colour of the blood is derived.

The black discolouration of the blood originating in this state of the capillary circulation is much more frequently observed in some organs than in others, and is seldom considerable, unless at an advanced period of life. There are, strictly speaking, only two organs, the respiratory and digestive, in which this change of the blood bears any resemblance to true melanosis.

In the lungs the black discolouration of the blood accompanies all those diseases which impede mechanically the capillary circulation, and render the function of respiration imperfect; such as phthisis pulmonalis, emphysema, chronic catarrh, dilatation of the bronchi, induration of the pulmonary tissue, and various diseases of the heart. The black discolouration may pervade the whole of the lungs, a limited portion of their tissue, or a multitude of spots or minute points. It is never so intense when general as when partial. It occurs not only much more frequently, but has a deeper tint in the summits than in any other portion of the lungs.

The blood in this state of discolouration constitutes the “*Matière noire pulmonaire*” of Laennec. It may exist either alone, or in combination with accidental products, and presents the three following forms, viz. the punctiform, ramiform, and maculiform. Of these three forms, the *punctiform* and *ramiform* are the most frequent in their occurrence. They may be observed in any portion of the pulmonary tissue, and may be either diffuse or circumscribed. When diffuse, the substance of the lungs presents a dark grey or slate blue colour, arising from the discolouration of the blood in the minute veins and capillaries. The vessels are best seen in the inter and intra-lobular tissue beneath the pleura. They are ramiform in the former, capilliform and punctiform in the latter. When circumscribed, the discolouration is most frequently accompanied with tuberculous matter, around which the bloodvessels in which the discoloured blood is contained assume a stellated arrangement, or appear like black points scattered throughout the minute divisions of the tuberculous matter. The grey or bluish grey colour of the tuberculous matter so often met with, is occasioned by the presence of these black points, which appear to have their seat in the air-cells, the circulation in which is early impeded by the accumulation of the tuberculous matter within them.

[*Melanoma*, 2.]

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The *maculiform* black discolouration of the pulmonary tissue is seldom met with except in the upper lobe of the lung in old persons. A portion,—most frequently the summit, or the whole of this lobe,—is often found in them studded with dark grey, purple, blue, and black spots. The pulmonary tissue contains little or no air, is firm and œdematous, or quite hard and somewhat dry. Masses of a putty-looking or cretaceous substance, of fibrous, cartilaginous, or osseous tissues, are generally seen lodged in the darker portions of it, and the bronchi and bloodvessels are either much compressed or obliterated. This kind of discolouration follows the cure of tubercular phthisis, and is obviously the consequence of the interrupted state of the venous circulation in the affected part of the lung, in which the congestion, coagulation, and discolouration of the blood that follow in succession, can easily be traced to this cause. The grey slate colour of the indurated pulmonary tissue which forms the boundaries of tubercular excavations is of the same nature as the preceding.

Although this form of black discolouration of the lungs may occur in masses of the size of a pea or bean, it is easy to distinguish it from melanosis, as it has never the globular arrangement which this disease presents when it affects these organs. In melanosis the pulmonary tissue, instead of being hard, is soft and crepitant around the melanotic tumour; and the tumour itself is soft and somewhat spongy compared with the black masses of pulmonary tissue, which are sometimes as hard as cartilage and of a very compact texture. Two or more isolated bronchial glands, or round masses of hard tuberculous matter when coloured black, are more likely to be taken for melanotic tumours, but the smooth compact texture of the one enclosed within its capsule, and the tubercular composition of the other, enable us to determine the real nature of both.

Black discolouration of the bronchial glands appears to occur under the influence of the same causes which give rise to it in the pulmonary tissue. It is most frequently seen and most marked in the bronchial glands of old people, but it is sometimes considerable in young persons, and even in children affected with tubercular phthisis. The black discolouration of these glands is accompanied in general with tuberculous matter, osseous and cretaceous substances contained within them, or a considerable degree of induration of their substance. The congestion and discolouration produced by these mechanical causes vary in extent and degree; sometimes only a few, at other times the whole of the bronchial glands being affected, studded only here and there with red, grey, and black points, or of a deep red, brown, or black colour throughout their whole substance.

The black discolouration of the blood from stagnation in the digestive organs, is confined to the villous and follicular structures of the mucous membrane. The appearances to which it gives rise in these situations are, as I have already remarked, so similar to those produced by the action of an acid on the blood, that they do not require to be described apart. As the black discolouration of the blood from stagnation, whether arising in chronic irritation or a mechanical obstacle to the return of this fluid from the intestines, can only be confounded with that produced by an acid chemical agent, I must again suggest the necessity of testing the contents of the affected portion of intestine, should any doubt arise as to the nature of the lesion which it presents.

With regard to the plates of the present fasciculus, it is necessary to observe that they do not contain representations of the black discolouration of the blood in the vessels of the stomach from the chemical action of the gastric acid. They have been reserved for the succeeding fasciculus, as they illustrate one of the forms of softening which takes place in the digestive organs.

DESCRIPTION OF THE PLATES.

PLATE I.

Fig. 1, true melanosis of the liver. AA, a section of a number of globular tumours, varying from the eighth of an inch to an inch and a half in diameter, of a deep brown or black colour, homogeneous aspect, uniform texture, of the consistence of a lymphatic gland, and lying in immediate contact with the substance of the liver. B, small tumours of a similar kind, situated under the pleura. CC and D represent appearances which are sometimes seen to precede the formation of these tumours. The deposition of the melanotic matter has taken place at C, in a multitude of points, giving an appearance to the substance of the liver of having been dusted over at that part with powdered charcoal. At D, the melanotic matter is contained in the minute veins, and presents, when narrowly examined, a beautiful ramiform distribution. The union of these points and venous branches is, as I have said, occasionally seen to produce the melanotic tumour. The appearances shown at D illustrate also the fact of the melanotic matter being contained in the blood of the venous capillaries, prior to its deposition in the molecular structure of the liver.

Fig. 2 represents melanosis of the lung. Several of the melanotic tumours, AAA, which present the same physical characters as those contained in the liver, are lodged in the substance of the lung; others, B, are situated beneath the pleura; several, CC, are attached to the free surface of the pleura pulmonalis; and others, DD, to that of the pleura costalis. All the tumours contained in the substance of the lung are globular, and vary from the size of a pin's head to that of a cherry. Those attached to the surface of the pleuræ are either globular or pyriform, the latter being pedunculated. We have likewise in this fig. at E, an example of the stratiform melanosis. The melanotic matter was quite fluid, but enclosed in fine cellular tissue, both of which could be removed from the surface of the pleura without abrading it. The tumours attached to the pleura appeared also to be formed by the deposition of the melanotic matter on the surface of this membrane, after the manner of secretion, for in some of them this matter was also in the fluid state, and enclosed in a fine serous capsule, which was obviously not a reflexion of the pleura, but a new formation, produced in the manner already explained.

Fig. 3 has been given as a fine specimen of melanosis of the skin, in which it has been said the disease does not occur. It is from the grey horse. A number of round

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melanotic tumours, AAA, of various sizes, single and grouped together, are situated in the subcutaneous cellular tissue; several, B, are seen embedded in the substance of the cutis vera; and others, DD, project above the surface of the skin, covered with white and grey hairs. In some of these tumours the melanotic matter was black, and in others, C, it was of the colour of bistre or umber.

Fig. 5 is also a specimen of melanosis from the horse. It represents one of those masses of aggregated tumours of the cellular tissue so common in this animal. AA, the tumours grouped together, and enclosed in a common cellular capsule. B, a section of one of these tumours, in order to show the deep black colour and pitchy aspect of the melanotic matter. C, the membranous capsule of the tumour partly dissected, shewing several bloodvessels ramifying upon it, but not passing into the melanotic matter. Similar vessels are seen on the surface of the large central tumour. D and E, a large artery and vein included within the general mass of tumours, and compressed.

Fig. 4 represents one of the simplest of those melanotic tumours, which I have denominated compound. It is composed chiefly of fibrous tissue, A, between the lamellæ and fibres of which the melanotic matter, of the colour of umber, is deposited. A strong cellulo-fibrous capsule, B, enclosed the whole tumour, and served as the basis of attachment to the fibrous tissue, from which a portion of the former, C, has been separated by dissection.

PLATE II.

This Plate contains seven figures, illustrative of a remarkable case of melanosis that occurred in an old man between seventy and eighty years of age, brought to the Hôtel Dieu of Paris in a state of incomplete paralysis, and incapable of giving any account of himself. He lived several days, had little or no fever or excitement of any kind. The paralysis soon became complete and general, and he died in a state of collapse and profound stupor. On examining the body after death, deep brown or black-coloured tumours, of various sizes, were found in several organs. The brain contained two in each hemisphere, as large as a hen's egg. *Fig. 1* represents a section of the right hemisphere, and two of these tumours, AA, situated at its basis, one of them towards the anterior, the other towards the posterior lobe. The latter had made its way through the walls of the right ventricle, poured a quantity of the black fluid with which it was impregnated into this cavity, E, which had passed from thence into the opposite ventricle, the third and fourth, and along the spinal cord to its inferior extremity. Although these tumours were almost entirely surrounded by the medullary substance, they must have been formed originally on the external surface of the brain, between the convolutions, for they were all intimately connected with the pia mater. Thus the two large tumours at the basis of the brain are covered, inferiorly, by this membrane, which is seen raised at D, by the tumour contained in the anterior lobe; and a small one, B, partly lodged between the cortical substance of a deep-seated convolution, is also covered externally by the same membrane. Besides, the bloodvessels of the pia mater passed into these tumours, and constituted by far the greater part of their bulk. The bloodvessels were crowded together at their exit from the pia mater, became tortuous in the substance of the tumours, some of them being nearly a line in diameter; were reflected backwards at their extremities in the form of irregular interlaced bundles, towards which

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two or three small arteries coming from the pia mater were seen to distribute themselves. The black colouring matter of these tumours was very abundant, nearly as fluid as ink, and was contained not only in the loose cellular tissue which separated the bloodvessels from one another, but likewise in the vessels themselves. The veins (or large tortuous vessels described above) were completely filled with it, whilst the arteries, minute as they were, were distinctly seen to carry red blood. The most of these appearances are represented in *fig. 2*, a section of the large tumour of the anterior lobe *in situ*, after having been deprived of a quantity of the black fluid matter by pressure and ablution, in order to render the structure of the tumour more conspicuous. A, the tumour surrounded by the medullary substance, which was reddened, congested, and somewhat soft. B, the veins and arteries passing from the pia mater into the tumour, and arranged in the manner described. The arteries are distinguished from the veins by the former being very slender and of a pale red colour.

Fig. 4 and *5* represent a tumour of the same kind formed in the epiploon. In *fig. 4*, the tumour, A, about the size of a walnut, is seen attached to a prolongation of the epiploon, B, between which and the tumour there existed the same kind of vascular connexion as between the pia mater and the tumours in the brain. It presented also the same internal structure as the latter, as seen in *fig. 5*, which represents a section of it after the removal of a quantity of its black fluid contents.

Fig. 6 represents a portion of the ileum from the same individual, containing several black tumours, of a round or oval shape, flattened externally, and varying from a quarter of an inch to an inch in diameter. They had all perforated the mucous coat; some of them, AAA, projected above the surface of the intestine, and the largest, B, formed an irregular ulcer, of considerable size. They were seated in the sub-mucous coat, as seen in *fig. 7*, which represents a section of one of the tumours, and the coats of the intestine. A, the tumour, situated between the mucous membrane, C, and the muscular coat, B.

The cortical substance of the brain contained three or four melanotic tumours, rather larger than hempseed, one of which is represented in *fig. 3*, A. They were quite unconnected with the pia mater, were not composed of bloodvessels like the others, possessed the consistence of a small lymphatic gland, and contained but a minute quantity of cellular tissue.

Besides these black tumours, vascular and non-vascular, there was one of another kind, about the size of a small cherry, situated in the brown substance of the corpus striatum of the right hemisphere. Instead of being black, it was of a reddish grey colour, *fig. 1*, C, and was traversed by a great many minute vessels filled with red blood. It was nearly of the same consistence as the others, but when pressed discharged a small quantity of a milky or creamy-looking fluid. After having been submitted to gentle pressure and ablution, it assumed an appearance somewhat similar to that observed in the black vascular tumours. Its tissue was of a spongy reticulated character, of a dull red colour, and vascular.

I have entered more minutely into the details of this case than would have been necessary had it been one simply of true melanosis. It is indeed a remarkable example of the compound melanotic tumour, that is to say, true melanosis combined with carcinoma and erectile tissue, and occurring under circumstances which enable us to explain the origin and mode of formation of this kind of tumour. The original state of the black vascular tumours appears to have been the same as that of the small tumour situated in the corpus striatum. The former, however, had, in consequence of their great develop-

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ment acquired much more conspicuously the cellulo-vascular organization of the erectile tissue than the latter, in which this structure was observed in combination with carcinoma. Into this tissue the melanotic matter was subsequently deposited, but instead of forming a homogeneous mass as it did in the cortical substance of the brain, where it presented all the characters of the tuberiform melanosis, it assumed the form and arrangement of the anatomical elements of the original tumours, that is to say, it was collected in the veins and cellular tissue of which these tumours were composed. This case furnishes us likewise with a striking example of the formation of the melanotic matter in the blood, for the veins of the vascular tumours were completely filled with this matter; and Lobstein relates a case of a similar kind, in which it was found both in the veins and arteries.

Fig. 8 represents a portion of the small intestine and omentum affected with melanosis, chiefly to show the difference between this disease and the black discolouration of the blood on the same parts, produced by the chemical action of the acid contents of the digestive organs. A, the intestine; B, the omentum, with the melanotic matter accumulated beneath the peritoneum, in the form of small, flat, round, or oval-shaped masses of a uniform deep black colour.

PLATE III.

Fig. 1, a portion of the liver affected with melanosis and carcinoma. The carcinomatous matter is much more abundant than the melanotic. The former is recognised by its pale white or yellowish grey colour, the latter by its brown or black colour. Both have assumed the tuberiform arrangement; for AAA are melanotic tumours; B, carcinomatous tumours of a small size. The structure of the liver is still conspicuous from the form and arrangement of the acini, but the colour of these bodies has disappeared, and their bulk is increased, owing to the proper substance of the liver being replaced by these accidental deposits. A considerable quantity of the carcinomatous matter is accumulated at C, and appears to be increasing in bulk by the aggregation and enlargement of the neighbouring acini, which are seen forming an irregular border along the inner side of the principal mass. The melanotic matter being in less quantity than the carcinomatous, appears as if it had succeeded the latter, and were deposited principally in the cellular tissue which separates the acini, this tissue containing more of the black matter than these bodies. This disposition of the melanotic matter is well seen at DD.

Fig. 2 represents the greater part of the upper lobe of one of the lungs, presenting the general appearance of the universal black discolouration of these organs from the inhalation of carbonaceous matter. AA, the cut surface of the lungs of a black colour, but some parts of a deeper black than others. Some of the bronchi and bloodvessels are seen divided, and are much less discoloured than the substance of the lungs. The external surface of the lungs has a dirty blue colour, but still presents traces of the lobular structure so apparent in the healthy state.

Fig. 3 is a representation of the black discolouration of the lung from stagnation of the blood in this organ. Both lungs were studded with irregular groups of firm tubercles, and presented throughout the same kind of discolouration as that seen in the figure. The tuberculous matter, AA, was of a grey colour, interspersed with black dots, lines, or ramifications of minute bloodvessels, and the pulmonary tissue in which the

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tuberculous deposition had not taken place, was of a nearly uniform dark-slate or bluish-black colour. The black discolouration of the blood in the vessels of the inter and intra-lobular cellular tissue, BB, is strongly marked on the external surface of the lung. The patient, a female, was only eighteen years of age.

Fig. 4, the same kind of black discolouration of the lung and bronchial glands from a mechanical obstacle to the return of the venous blood, produced by cretaceous, fibrinous, and cartilaginous substances contained in these organs, and also from chronic induration of their respective tissues. AA, the summit of the upper lobe of the lung, the structure of which is entirely effaced by the presence of grey, blue, purple, and black patches of various sizes, amongst which are seen irregular portions of the accidental substances mentioned above, obliterated bloodvessels and bronchi. The whole of this part of the lobe felt as hard as cartilage. Inferiorly at B, the induration was not so considerable; the pulmonary tissue was somewhat granular, and here and there of a slight reddish tinge, œdematous and still crepitant,—appearances which indicated a less interrupted state of the circulation in this than in the upper portion of the lobe. The whole of the upper lobe was covered by a fibrous membrane, C, which held the two pleuræ in close contact, and which, by preventing the full expansion of the lungs, must have contributed to retard the circulation of the blood through these organs. The bronchial glands, as well as those situated along the inferior portion of the trachea and œsophagus, presented the same discolouration of the blood as that observed in the lungs. The glands EE, situated around the trachea D, were but slightly congested or of a light greyish blue tint; while those at its bifurcation, F, were gorged with blood of a deep red, purple, or black colour. Some of them, G, were greatly enlarged, very hard, contained a putty-looking or cretaceous substance, and were throughout of a reddish grey, deep blue, or black colour. The patient was upwards of fifty years of age.

Fig. 5 and *6* represent those appearances produced by the black discolouration of the blood in the villous and follicular structures of the intestines, either from stagnation of this fluid, or from its having been submitted to the action of an acid. In *fig. 5*, A indicates one of the glands of Peyer enlarged, and rendered very conspicuous by the black discolouration of the orifices of the individual follicles of which it is composed. BB and CC, the isolated follicles in which the black discolouration of the blood has taken place around the orifices of some, the basis and orifices of others, and has produced the punctiform and annular discolourations which I have already described. *Fig. 6* shows the *couleur ardoisée*, or slate colour of the mucous membrane, of French pathologists, which consists in the black discolouration of the blood in the capillaries of the villousities, in consequence of chronic inflammation, or the chemical action of the acid contents of the intestine.

PLATE IV.

This plate exhibits several forms of black discolouration of the blood from the chemical action of the healthy gastric acid, fluid, and gaseous acid products formed accidentally in the intestines.

Fig. 1 represents the fundus, A, of the stomach, laid open and suspended, filled with blood, B, deprived of its red colour by the gastric acid. The blood in this state resembled a thick solution of bistre or China ink. It was in great abundance in the stomach, and a considerable quantity of it was vomited two days before the death of the

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patient. The source of the sanguineous effusion was follicular ulceration, from an attack of sub-acute gastritis occasioned by indigestion. The stomach was previously in a state of disease, that is to say, the mucous membrane was very much thickened, and the follicles so much enlarged as to be distinctly seen over the whole surface of the stomach. CC and DD represent the enlarged mucous follicles, and the central ulceration whence the blood was effused.

Fig. 2, the stomach of a child laid open in the direction of the small curvature. A the pyloric, B the cardiac orifice, C the fundus of the stomach. Hemorrhage had also taken place in this case in the situation of the mucous follicles, but these glands were not enlarged, and the blood, instead of being effused into the cavity of the stomach, was accumulated in the mucous and sub-mucous tissue in the form of petechiæ. These petechiæ are of a deep brown or black colour along the great curvature and fundus of the stomach, DD, where the blood was exposed to the chemical action of the gastric acid; and red, EE, at the pylorus, cardia, and small curvature, where this fluid had not been submitted to the chemical influence of this agent.

Fig. 3, a portion of the colon from a person who had suffered long from dysentery, death having been preceded by a severe attack of hemorrhage. The mucous and sub-mucous coats were greatly thickened and extensively ulcerated. The mucous membrane was considerably raised around the ulcers, AAAA, and these were separated by deep irregular fissures. In some parts the mucous and sub-mucous coats were completely destroyed, BB, around the whole circumference of the intestine. The blood poured into the cavity of the intestine was converted into a black fluid resembling liquid pitch, and the ulcers themselves were impregnated with blood similarly altered.

Fig. 4 represents the chemical discolouration of the blood when effused beneath or on the surface of the peritoneum, and which by some pathologists has been confounded with true melanosis. In this figure the gradual discolouration of the effused blood from the action of the acid contents of the intestine is well seen. The degree of discolouration of the blood is obviously in proportion to the length of time it has been effused. At A the effusion is recent, and the blood is only beginning to lose its red colour; at B and C it changes from dark brown to black, apparently in the successive order of its effusion. This form of the chemical discolouration of the blood on the peritoneal surface is what I have named the *Stratiform*.

In *Fig. 5* we have a good example of the *ramiform* discolouration of the blood from the same cause, and in the same situation. I have described it as occurring in conjunction with chronic tubercular peritonitis. In this case the tubercles were very small, and many of them were nearly obscured by the discoloured blood accumulated in the vessels which ramified around them. The stellated arrangement of the vessels is well marked at AA, in the centre of which is perceived a small grey tubercle. At B is another form already alluded to, in which the black discolouration succeeds to ecchymosis. The blood in several of the mesenteric veins, CC, partakes of the same black discolouration as that observed in the vessels situated around the tubercles. If these two last figures are compared with figure 8, Plate II. the difference between true and spurious melanosis on the peritoneal surface will be at once apparent.

Plate I.

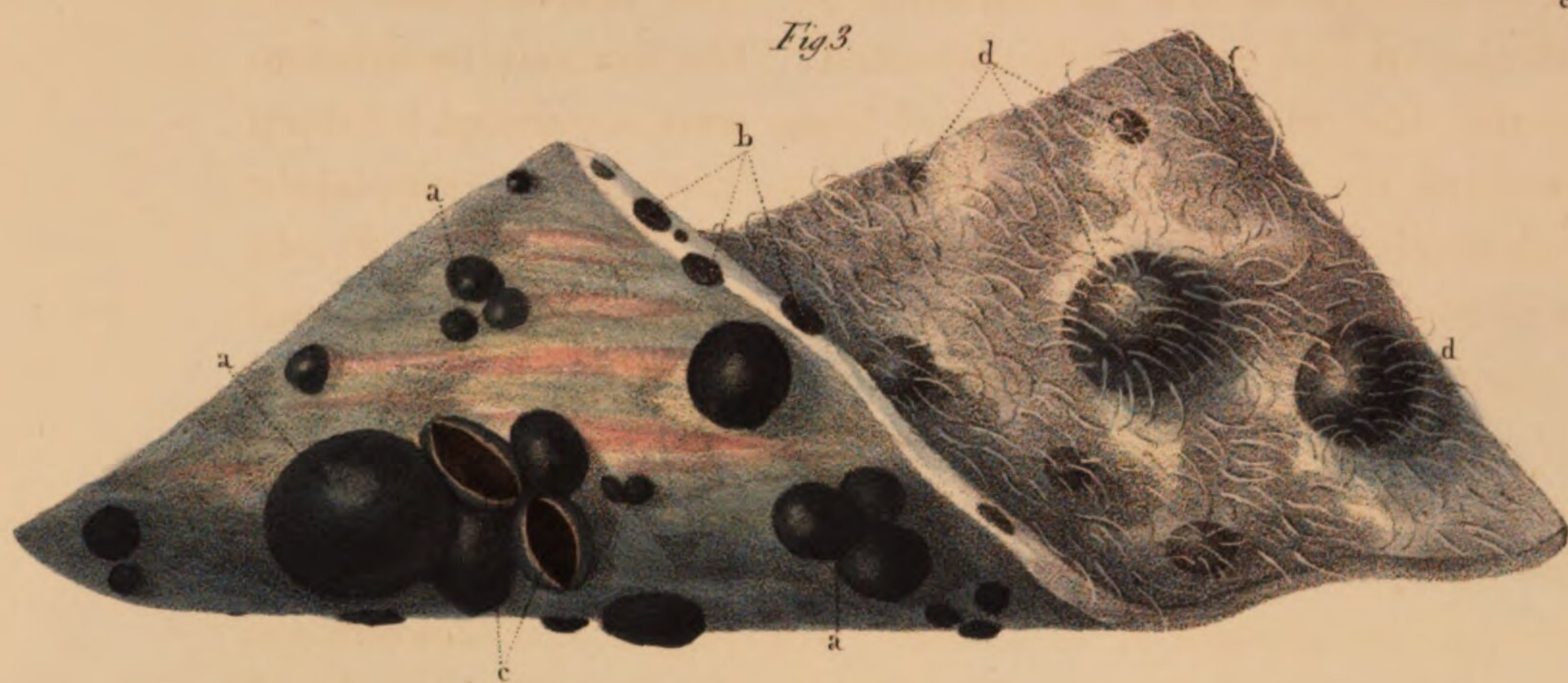


Plate II.

Fig. 1

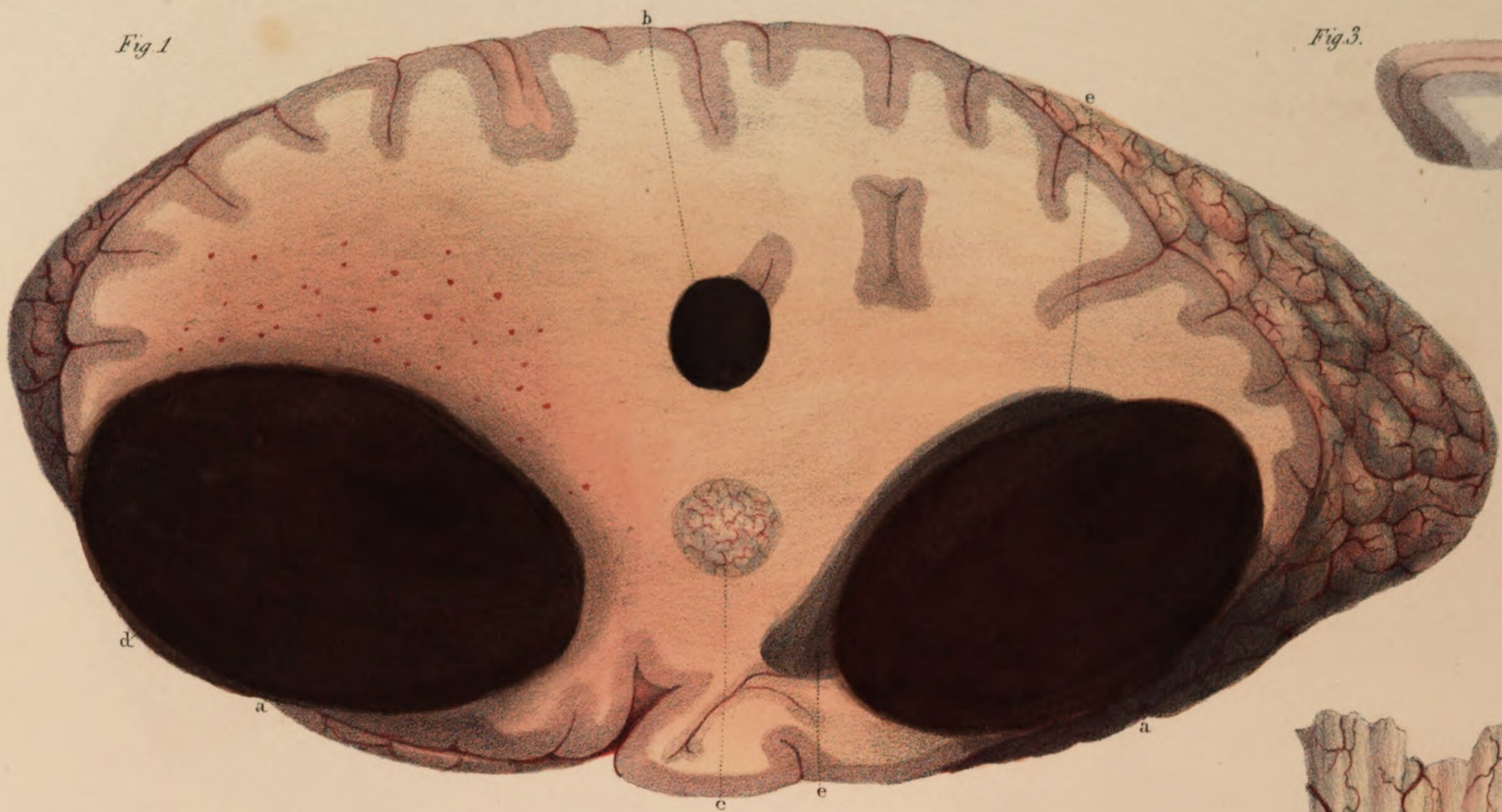


Fig. 3.



Fig. 2.



Fig. 5.



Fig. 7.



Fig. 4.



Fig. 6.

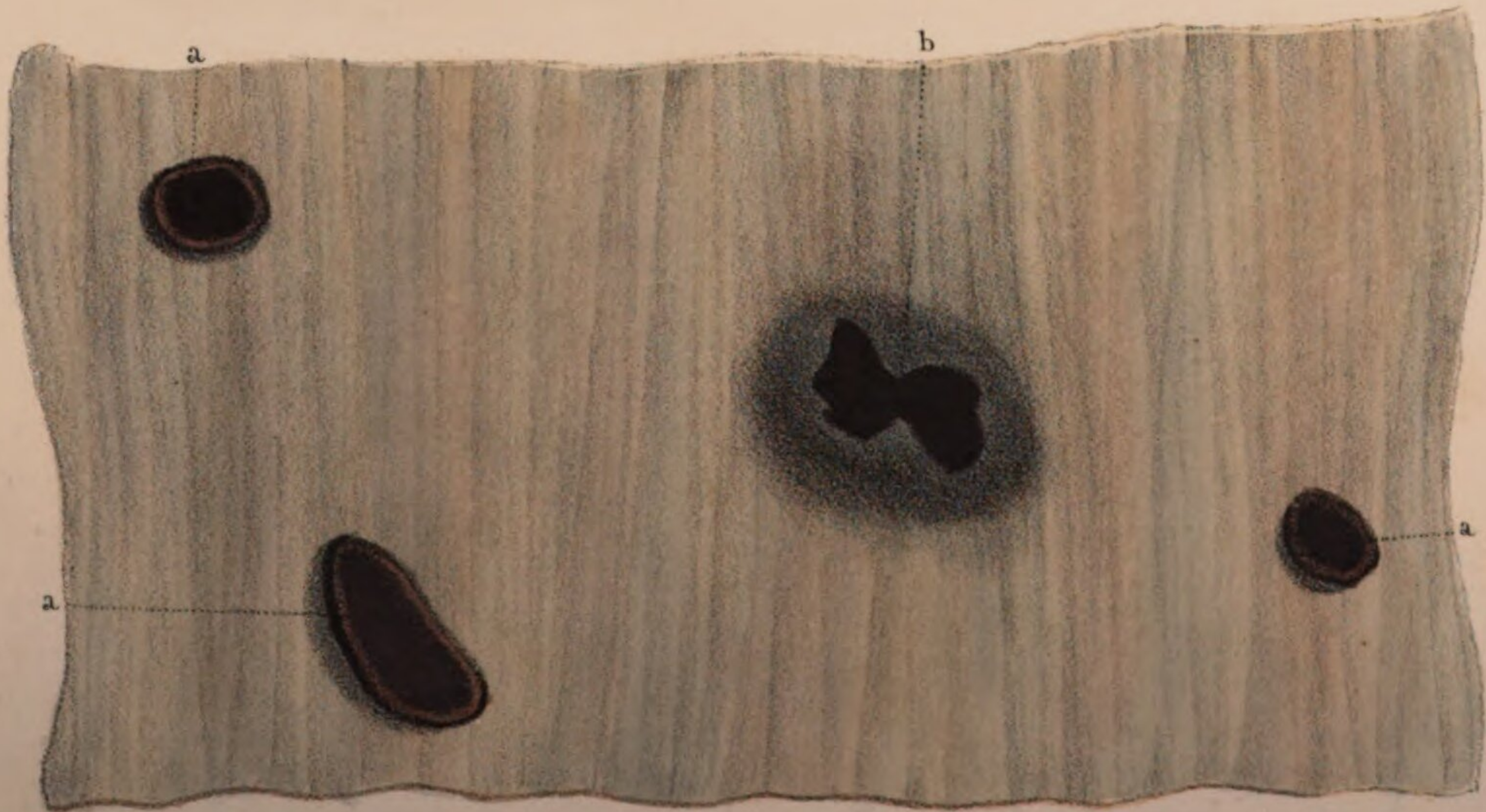


Fig. 8



Plate III.

Fig 2.



Fig 1.



Fig 3.



Fig 4.

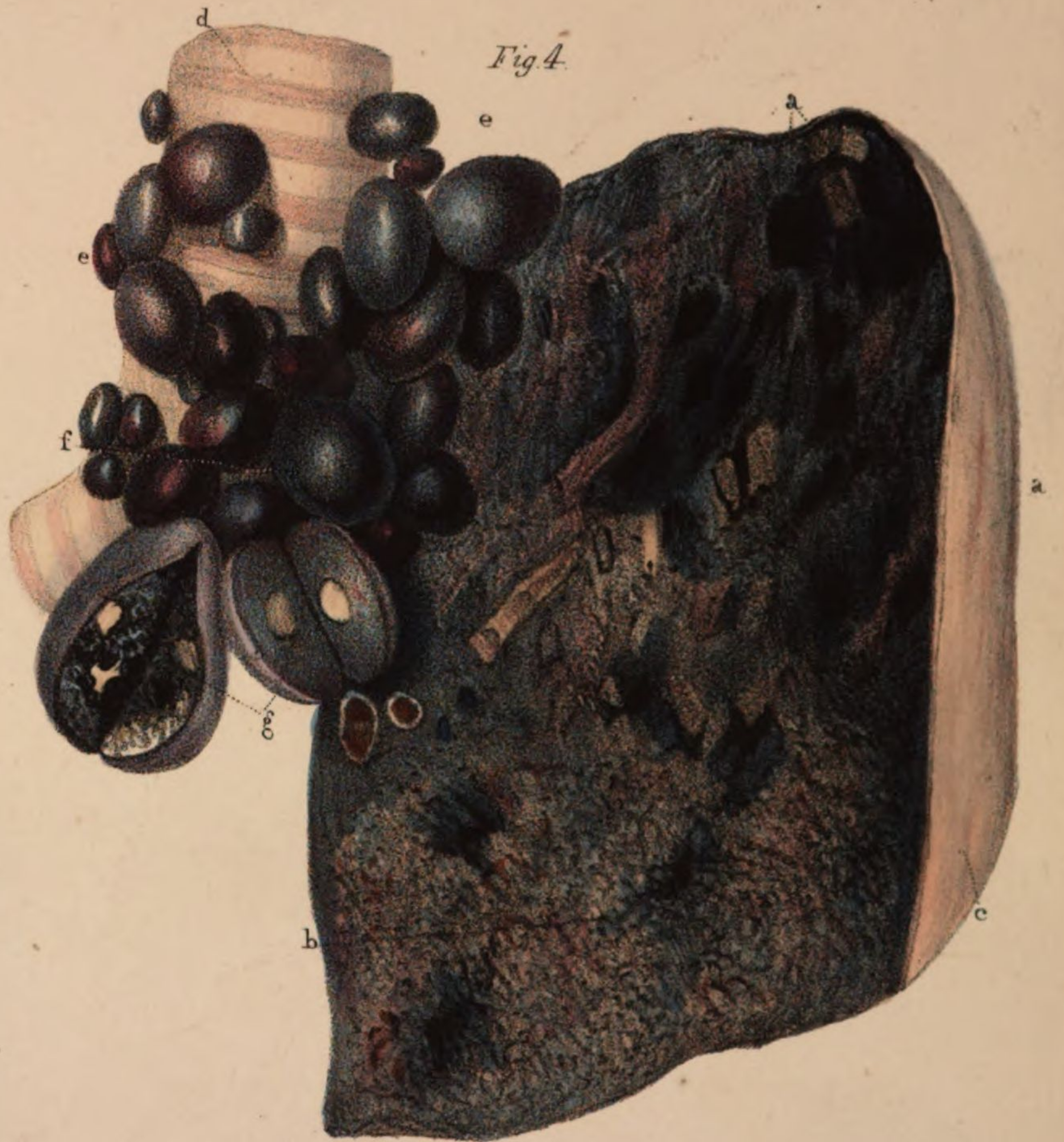


Fig 5.

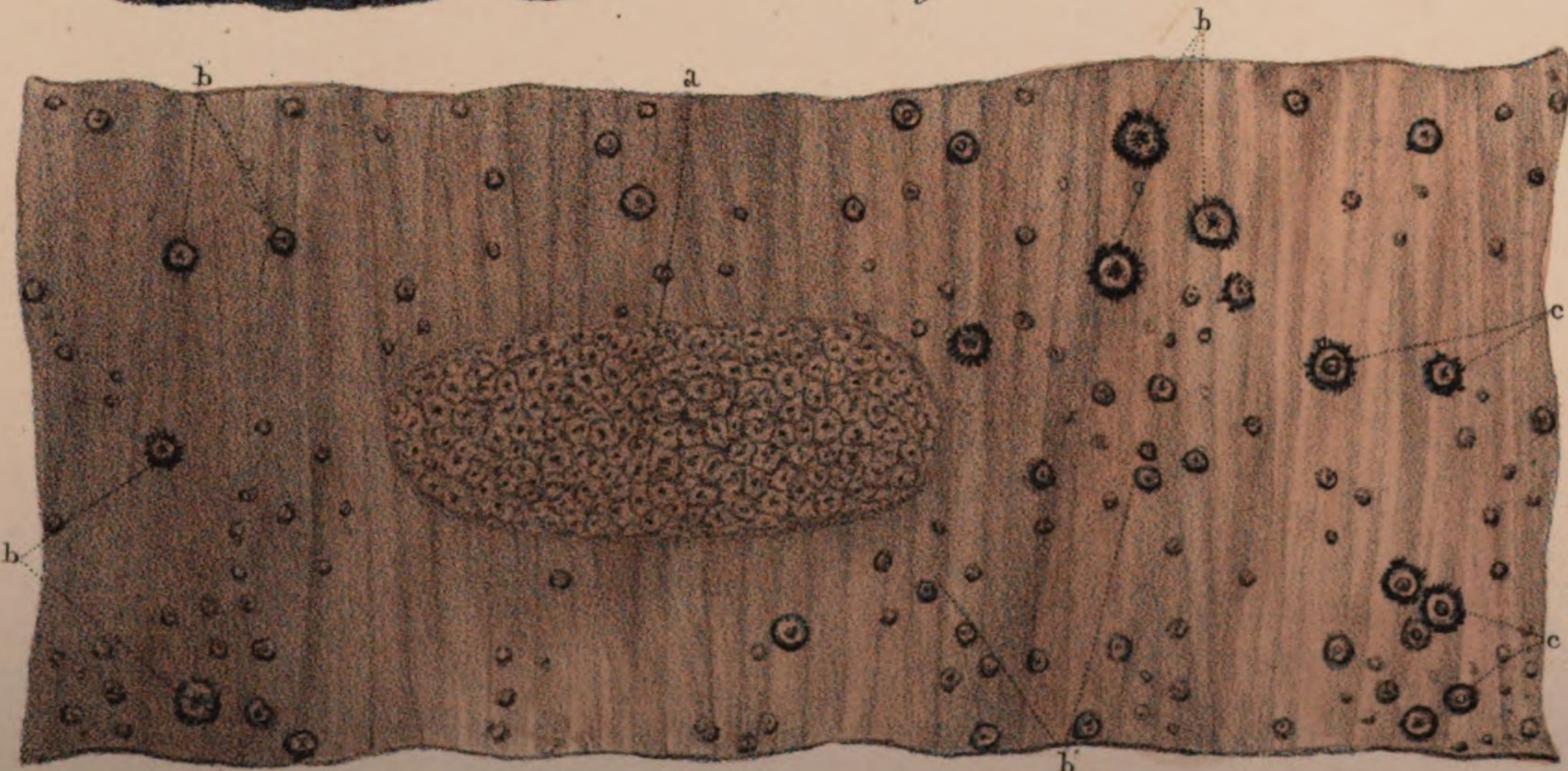


Fig 6.



Plate IV.

Fig. 4.



Fig. 3.



Drawn on stone by J. Carrwell

Fig. 1.

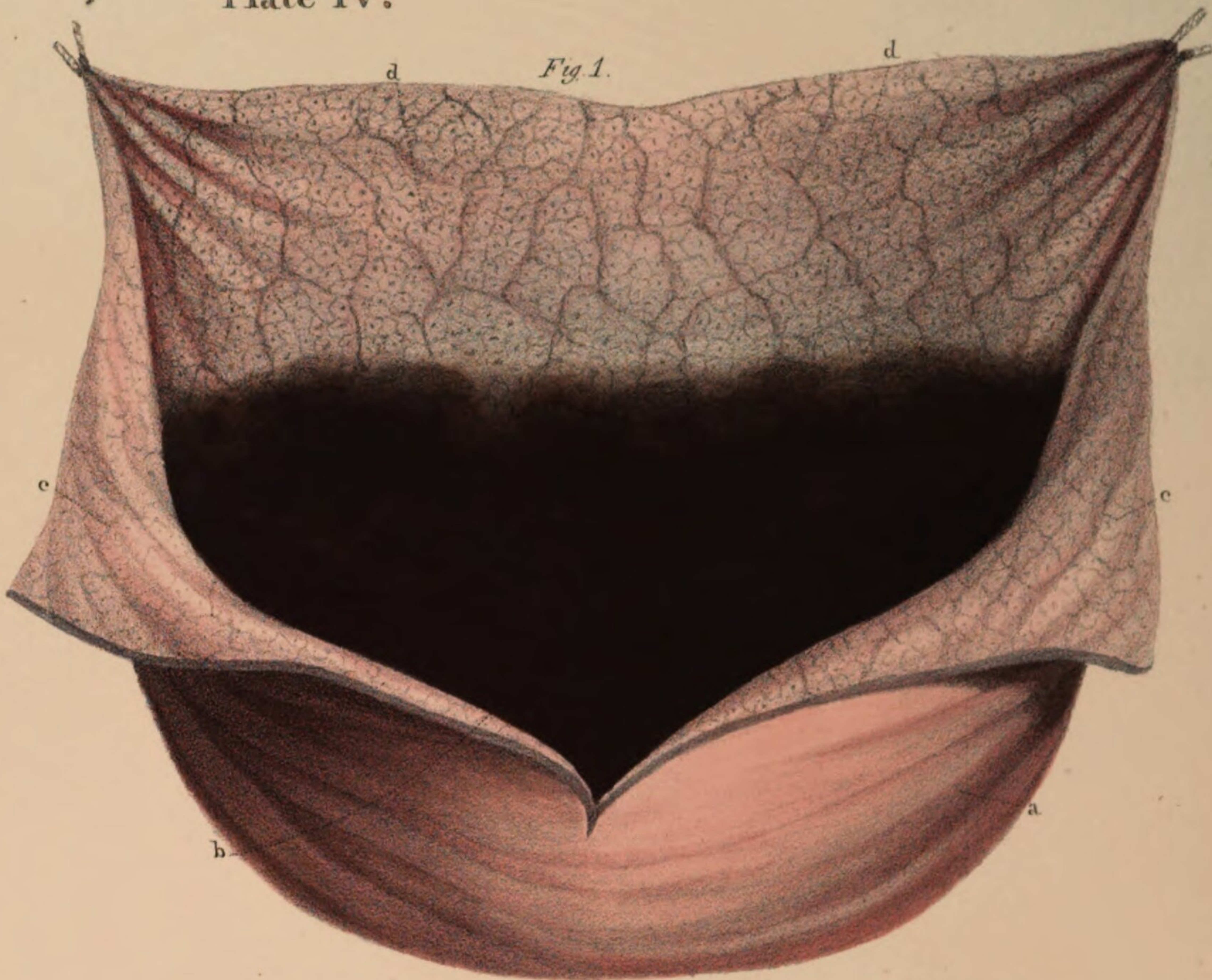


Fig. 2.

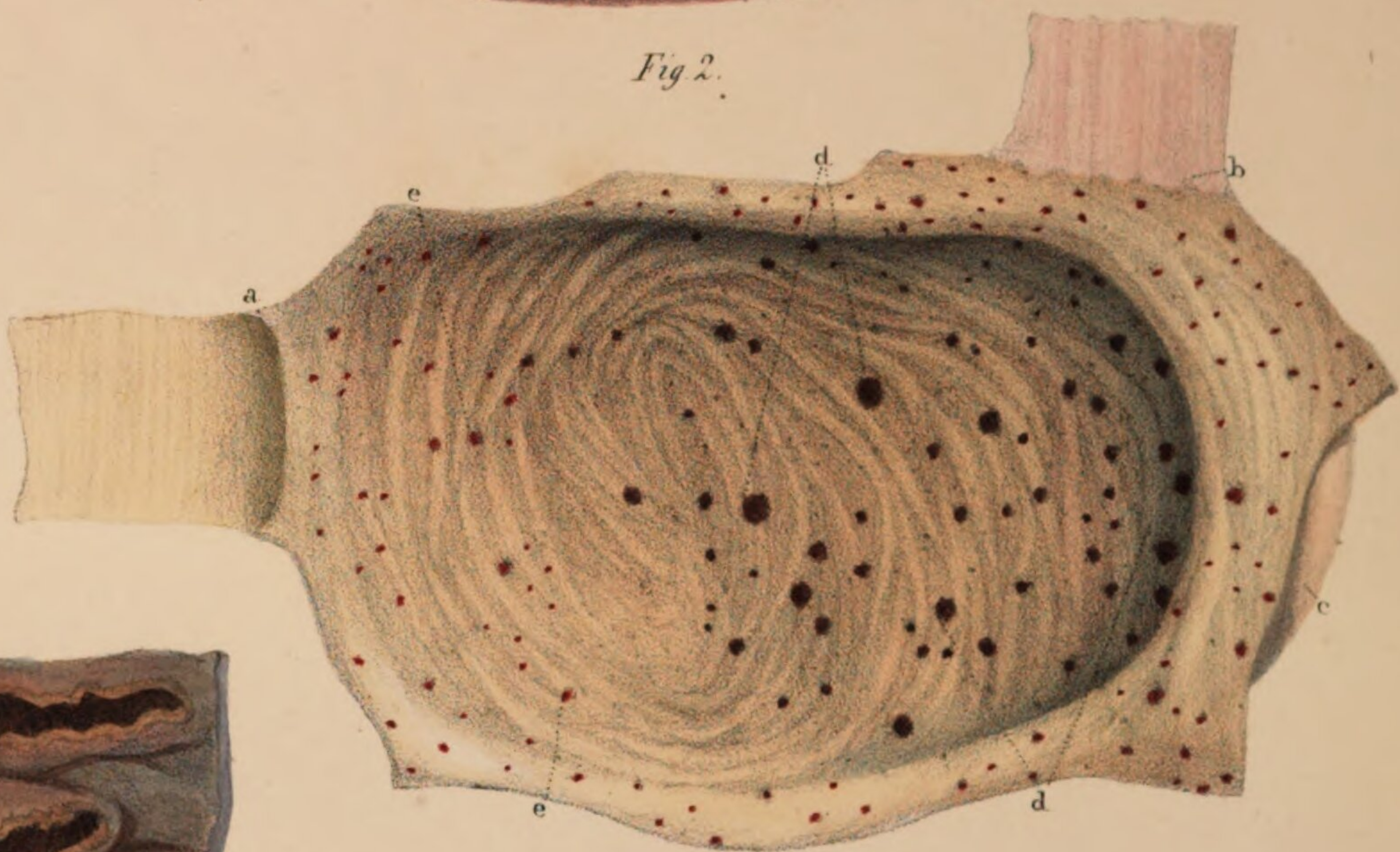


Fig. 5.



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C A R C I N O M A .

HETEROLOGOUS FORMATIONS.

CARCINOMA.

SEVERAL of the diseases comprehended under the title of Heterologous Formations constitute the most important, and, at the same time, most difficult subjects of anatomico-pathological investigation. Although they have long occupied the attention of pathologists, our knowledge of them is, in many respects, still extremely imperfect, particularly as regards those which have received from the surgeon the appellation of *malignant*—as cancer, medullary sarcoma, and fungus hæmatodes.

The imperfect state of our knowledge of these diseases may, I am inclined to believe, be attributed to the following circumstances, viz. the almost exclusive study of them for a long period as they occur in the external parts of the body in the form of tumours, and the partial or inaccurate observation of their primary forms, of the subsequent changes which take place in them, and of the modifications which they undergo in these and other respects, in the different tissues and organs of the body. These important circumstances have not, certainly, been altogether overlooked by some recent pathologists; but it does not appear to me that they have taken advantage of them, either to establish the distinctive characters of these diseases, or to explain the mode of their production. Besides, a multitude of names have been given to the same disease, on account of its having been examined by different individuals at different periods of its development, or in different organs; and hence an additional source of difficulty to those who have made the Heterologous Formations the subjects of their researches, as well as of the confusion that pervades the nomenclature of these diseases. It is, therefore, not surprising that the attempts which have been made to classify the Heterologous Formations have not been very successful. As the limits and objects of this work do not permit me to give an exposition of the several classifications that have been proposed, I shall, when describing these formations, allude particularly to such classifications of them as possess a scientific character, as well as to the principles on which they have been founded.

General division and arrangement of the Heterologous Formations.—The essential character of the Heterologous Formations consists in the presence of a solid or fluid substance, different from any of the solids or fluids which enter into the healthy composition of the body. This substance, however, presents such a marked difference in regard to one or more of the characters by which it is recognized, as at once to lead us to regard it as differing in its nature, and therefore constituting diseases of different kinds. It may resemble cartilage, brain, blood, the pigmentum nigrum, soft cheese, cream, an earthy concretion, or a stone; and hence, from the greater or less degree of resemblance which it bears to one or other of these natural products, it has been denominated scirrhus, medullary sarcoma, fungus hæmatodes, melanosis, tubercle, pus, and calculus.

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However, these terms are by no means to be regarded as implying a specific difference in the morbid conditions to which they have been applied. On the contrary, we shall find that the same disease has received various names, on account, as I have already said, of the great difference which it sometimes presents at different stages of its development; or different diseases are included under the same general denomination, because of their always presenting one common character.

To determine the number, and fix the distinctive characters of the diseases comprehended under the head of Heterologous Formations, it is obvious that we must, in the first place, determine the number and fix the distinctive characters of the latter, or rather of that peculiar substance which constitutes the essential character of these formations. This can only be done by making ourselves acquainted with the primary forms which this substance assumes, and the subsequent changes which it undergoes in the different tissues and organs of the body. It is, indeed, by a careful and accurate investigation of these latter circumstances that we may hope to accomplish the important objects in question, and to remove at least some of the difficulties which still attach themselves to the study of the Heterologous Formations.

Founding, as I have done, the essential character of the Heterologous Formations on the presence of a substance which does not enter into the healthy composition of the body, the several kinds of these formations may also be established on the same principle; the differences which they present being derived from certain characters which may be regarded as the type of each. They will thus form so many genera, the species and varieties of which will be determined with greater or less facility, according as they approach to or recede from that state which constitutes the distinctive character of the genus to which they belong.

Acting on these principles, I shall include under the following denominations what I conceive to be distinct diseases belonging to the class of Heterologous Formations, viz. Carcinoma, Melanoma, Pyonoma, Tyroma, Lithoma.

Specific division of Carcinoma.—In the genus Carcinoma I propose to comprehend those diseases which have been termed scirrhus; common vascular or organized sarcoma; pancreatic, mammary, and medullary sarcoma; and fungus hæmatodes.

The following reasons may be assigned for thus grouping together under the generic term of Carcinoma, so many diseases generally described as differing widely from that which is commonly known by this designation. 1st. They often present, in the early periods of their formation, certain characters common to all of them, however much they may differ from each other in the subsequent periods. 2d. They all terminate in the gradual destruction or transformation of the tissues which they affect. 3d. They have all a tendency to affect several organs in the same individual. 4th. They all possess, although in various degrees, the same reproductive character.

Such are the more remarkable phenomena which, considered anatomically and in a general point of view, these diseases present in common with one another, and which, I conceive, express in a conspicuous and important manner, that near relationship which justifies their being brought together under the same general denomination.

When we consider these diseases more in detail, we find that they present differences, some of which are of considerable importance, others much less so; and therefore it becomes necessary to separate them into distinct groups, and to arrange them into species and varieties.

The differences to which I allude are referable to two states of the Heterologous Deposit to which the diseases in question owe their origin. The first is that

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in which this deposit has little or no tendency to become organized. Its form and arrangement appear to be determined chiefly by external circumstances; and its formation and subsequent increase are entirely dependent on the nutritive function of the organ in which it is contained. In the second state this deposit exhibits, on the contrary, a greater or less tendency to become organized. Although it may, at first, assume a determinate form and arrangement, in consequence of the influence of external circumstances, it possesses in itself properties by means of which its subsequent arrangement and development are effected, independent of the nutritive function of the organ in which it is formed, except in so far as the materials of its growth may be derived from this source.

On account, therefore, of these two opposite states of the heterologous deposit, Carcinoma may be divided into two species, the first of which I shall call Scirrhomia, the second Cephaloma. Although neither of these terms expresses the essential characters of the respective states to which they are applied, I have not been able to find or devise others better calculated to accomplish this object. It is, therefore, necessary to bear in mind that they indicate only one of the characters of these states, viz. a certain degree of consistence, and which, it may be observed, is far from being constant in either, because of various circumstances which I shall afterwards endeavour to explain.

In these two species of Carcinoma, the Heterologous Deposit presents itself under various forms, which may be regarded as constituting so many varieties of each species.

Varieties of Scirrhomia.—The varieties of scirrhomia are determined chiefly by the relative quantity of the Heterologous Deposit, the manner in which it is distributed, and the difference of colour and consistence which it presents. Thus, it may be collected in numerous points in the form of a hard, grey, semi-transparent substance, intersected by a dull white or pale straw-coloured fibrous or condensed cellular tissue, and, as such, is commonly denominated *Scirrhus*. When it assumes a regular lobulated arrangement, so as to represent an appearance similar to a section of the pancreas, it forms what was called by Mr. Abernethy the *Pancreatic Sarcoma*. Again, it may be disseminated uniformly throughout the texture of an organ, which it converts into a solid substance resembling a slice of raw or boiled pork, and is then called by the French the *Tissu Lardacé*. Lastly, when it presents the appearance of firm jelly, and is collected into masses of greater or less bulk in a multitude of cells, it is the *Matière Colloïd* of Laennec, the *Cancer Gélatiniforme* or *Aréolaire* of M. Cruveilhier.

Varieties of Cephaloma.—The principal varieties of cephaloma are derived from the appearances which the Heterologous Deposit presents either in different organs or at different stages of its development. When it presents the appearance of firm coagulable lymph, or fibrine deprived of the red colouring matter of the blood, possessing a uniform, fibriform, or lobuliform arrangement, with a certain degree of transparency and vascularity, Mr. Abernethy gave to it the name of *Common Vascular* or *Organized Sarcoma*. In this state the Heterologous Deposit is generally collected into a mass of greater or less bulk, in which few or no traces of the proper tissue of the organ in which it is contained are observable. If, on the contrary, it be uniformly disseminated throughout the texture of an organ, so as to transform it into a substance resembling a section of the mammary gland, or the udder of the cow when boiled, the appellation of *Mammary Sarcoma* was given to it by Mr. Abernethy. When it presents an appearance similar in colour and consistence to the substance of the brain, it was called *Medullary Sarcoma* by the same distinguished surgeon; *Matière Cérébriforme* or *Encephaloïde* by Laennec; *Spongoid Inflammation* by Mr. Burns.

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The *Milk-like* tumour of Dr. Monro; the *Soft Cancer* of various authors; and the *Pulpy* testicle of Dr. Baillie, are names which have been given to the same state.

Of all the varieties of cephaloma, the last is that in which a vascular organization is most conspicuous; and as the coats of the vessels with which it is supplied are remarkably delicate, the circulation of the blood through them is readily interrupted; hemorrhage from congestive rupture takes place, and the effused blood is mixed in greater or less quantity with the brain-like matter. From this accidental circumstance, together with the protrusion of this substance through the ulcerated integuments for example, in the form of a bleeding fungus, it has been described by Mr. Hey and also by Mr. Wardrop, under the appellation of *Fungus Hæmatodes*. Sir Astley Cooper calls it *Fungoid Disease*.

Such are, in my opinion, the principal varieties of scirrhus and cephaloma, and the general characters by means of which they may be recognized. But although I have said that the essential difference between these two species consists in the former having little or no tendency, the latter a greater or less tendency, to become organized, it is by no means always easy, nay it is sometimes impossible, to draw a distinct line of separation between them; for the Heterologous Deposit when first formed, and indeed frequently for a considerable time after its formation, does not furnish us with any signs which show that it will or will not become organized. We cannot determine what those properties are by means of which it is afterwards to assume a definite arrangement, or to possess within itself the powers of contributing to its subsequent development. These facts may be illustrated by the two following circumstances:—1st. The Heterologous Deposit may exist as I have described it, in the form of scirrhus, pancreatic sarcoma, or the lardaceous tissue, without its presenting any trace of organization; the textures which it invades being gradually destroyed by its presence, and both ultimately converted into a soft granular, pulpy, or liquid mass, of the colour and consistence of cream or milk. 2d. The same deposit may exist under the same forms as those I have just named, but it undergoes changes of an entirely opposite kind; that is to say, it assumes the characters of the mammary or medullary sarcoma, becoming more or less soft and vascular, and frequently terminating in hemorrhage by the rupture of its vessels, or in that state called fungus hæmatodes.

While, therefore, the distinctions which I have drawn between the two species of carcinoma are founded in fact, the circumstances which I have just stated show at once the difficulty of laying down fixed rules for the discrimination of each, and at the same time justify that part of my arrangement which brings them together under the same general denomination.

Numerous examples might be given of scirrhus, medullary sarcoma, and fungus hæmatodes, as they are commonly called, originating in the same morbid state, and passing successively from the one into the other in the order in which I have named them. Indeed, we often meet with all the varieties which I have enumerated of both species, not only in different organs of the same individual, but even in a single organ. And of so much importance does it appear to me to establish this fact, that the coloured representations of carcinoma in the present fasciculus have been given almost exclusively with this view. If such are the successive changes observed to take place in carcinoma, the distinction of the disease into species and varieties may be considered by some to be of little importance. Such, however, cannot be the case; for we know that the curability of a disease often depends on the time at which a remedy is employed, or varies with the state or period of the disease; and, therefore, it is obvious

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that, whatever means may be adopted for the cure of carcinoma, we can form no precise opinion as to their relative efficacy, unless the particular condition or variety of the disease be kept in view. And it is far from being an unimportant fact that several of the varieties of both species of carcinoma differ materially from one another as regards the comparative rapidity of their development, as well as their reproductive tendency. Thus, in both these respects the pancreatic differs from the lardaceous, the lardaceous from the mammary, and the mammary from the medullary sarcoma; the first often remaining stationary for a long space of time, months or years; the last frequently acquiring its maximum of bulk in a few weeks, and, when removed, being sometimes reproduced with a degree of rapidity which is never observed in any of the other varieties. It may be said that the more the varieties of both species of carcinoma partake of the characters of the Analogous Formations, viz. the cellular, cellulo-fibrous, and fibrous tissues, they are, *cæteris paribus*, the less rapid in their development, and the less is their tendency to be reproduced.

Definition of Carcinoma.—From the general considerations into which I have entered on the species and varieties of carcinoma, it must be obvious that no precise definition can be given of this disease. It may, however, be said to consist in the formation or deposition of a peculiar substance, which presents great variety of consistence, form, and colour; frequently assumes a definite arrangement, and possesses a vascular organization of its own; gives rise to the gradual destruction or transformation of the tissues in which it is situated; affects successively or simultaneously a greater or less number of organs, and has a remarkable reproductive tendency.

Seat and origin of Carcinoma.—Various opinions have been entertained regarding the seat and origin of carcinoma, some of which have attracted notice merely on account of their novelty; others from their possessing much higher claims to our consideration, and to these I shall more particularly allude as I proceed.

There are several organs subject to carcinoma, which, from the peculiarity of their structure or other circumstances, afford us the means of ascertaining the precise seat of this disease, its origin, and mode of formation. But to derive all the advantages which these circumstances are capable of affording, it is necessary to catch, as it were, the disease at the earliest period of its formation, that is to say, when the heterologous substance of which it consists has just been deposited, and has not effaced the particular texture or structure of the part in which it is contained. Investigated in this its first stage, we ascertain with greater or less facility that this substance becomes manifest to our senses, either as a production of nutrition or of secretion. In the former case it is deposited in the same manner as the nutritive element of the blood, enters into the molecular structure, and assumes the form and arrangement of the tissue or organ into which it is thus introduced. In the latter it makes its appearance on a free surface, after the manner of natural secretions, as on serous surfaces in general.

Such are two of the modes in which the heterologous deposit which gives rise to carcinoma is formed, and also two of the principal differences in regard to its seat. Proceeding still farther in our researches, we find this substance existing not only in the molecular structure and on the free surface of organs, but also in the blood. But before proceeding farther in this important inquiry, I shall first endeavour to show in what manner this substance is deposited in the structure of organs.

Formation of Carcinoma in the molecular structure of organs.—The liver and stomach,—organs in which carcinoma is of frequent occurrence, afford us the most

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striking examples of the mode of formation of the disease. If we make a section of a liver containing what are commonly denominated carcinomatous tumours, that is to say, round or irregular masses of a substance resembling one or more of the varieties of scirrhus and cephaloma, we shall often be able to detect the first stage of these tumours, and discover the manner in which they are formed. Thus, the first thing which I have frequently been able to perceive in those portions of the liver in which the heterologous deposit is just making its appearance, is a slight change of colour, observable within a very limited and well defined space, and which is distinctly seen to exist in those minute divisions of this organ denominated *acini*. This change of colour may take place in a single acinus, or in several of these bodies successively or simultaneously. The red or yellow colour which they naturally present gradually disappears, and is succeeded by a pale milk-white or straw colour, accompanied by an increase of their consistence. But the most important circumstance is, that while these changes of colour and consistence are taking place, the form and bulk of the acini remain unaltered. Now it need hardly be mentioned that the form and bulk of the acini could not remain unaltered unless the foreign substance, to which their change of colour and consistence must be ascribed, were introduced into them in the same order as the normal element of nutrition,—unless it were deposited in the molecular structure of the acini, in a manner precisely similar to that in which their nutritive function is carried on. Otherwise, along with the change of colour and consistence which they present, we should have a simultaneous increase of their bulk. The more we examine the acini in this state, the more will we be persuaded that they are thus *transformed* by the molecular deposition of that peculiar substance which constitutes the essential anatomical character of the disease in question.

By tracing the transformation of the acini from a lower to a higher degree, we can perceive them forming groups, for example, of three, four, ten, or twenty, the reunion of which constitutes tumours varying from the size of a hemp-seed to that of a cherry, in all of which the structure of the liver as characterized by the form, bulk, and arrangement of the acini, is more or less conspicuously marked, but becoming gradually less so with the increasing bulk of the tumours, until it entirely disappears, being transformed into a uniform, lardaceous mass, or into some one or other of the tissues or substances which belong to either of the two species of carcinoma.

I have said that a similar mode of formation of carcinoma is observable in the stomach. It is, however, chiefly in the muscular coat of this organ that it is conspicuously seen, on account of the difference of colour between the muscular substance and the cellular tissue which enters into its composition. The change of colour which accompanies the presence of the heterologous deposit in this tunic is hardly perceptible except in the muscular fibres. These, however, become pale, and acquire an increase of consistence; but their bulk does not appear to be increased at first, and they retain their form and distribution. Such, also, is the state of the intermuscular cellular tissue at the same period, except, as I have said, as to colour, which is not sensibly changed on account of its being naturally pale. By-and-by both acquire a greater or less increase of bulk, become remarkably distinct, and present that fibriform arrangement, hardness, and transparency which are regarded as so characteristic of scirrhus. At a more advanced period of the disease we no longer trace this nutritive process of transformation; the muscular and cellular tissues being converted into a homogeneous mass, which is afterwards softened down, or assumes the mammary, medullary, or hæmatoid forms of

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carcinoma. The production of this disease in the molecular structure of organs will be more fully illustrated by means of the coloured representations contained in the present fasciculus.

Formation of Carcinoma on serous surfaces.—The fact of carcinoma forming on the free surface of serous membranes is strongly corroborative of the accuracy of the preceding remarks ; for although in the former case I have referred the presence of the Heterologous Deposit to a modification of nutrition, and in the present to a modification of secretion, the difference is merely nominal. The distinction, however, between nutrition and secretion is valuable as regards the formation of carcinoma ; for considered as a modification of the latter, we possess ourselves of the advantage of studying this disease under perhaps the most simple form which it presents, viz., on extensive serous surfaces, such as the pleura and peritoneum. Here the heterologous material is found to be effused on the free surfaces of these membranes, without our being able to perceive that they have undergone any previous change whatever. Multitudes of tumours are sometimes met with on these two surfaces, varying in bulk, consistence, and colour. Some of them are as large as a plum or an orange ; others of the size of cherries or peas, and composed of a substance resembling pork, the mammary gland, brain, or a mixture of the latter, fibrine, and blood. Scattered among these larger masses are a number of smaller bodies, some of them so minute as hardly to be perceived by the naked eye ; others varying from the size of a pin's head to that of millet or hemp-seed. They present the same characters as the former ; even the smallest of them may be nearly as firm as cartilage or as soft as brain ; pale, red, or bloody. Under such circumstances the source of these heterologous productions in the blood, their separation from this fluid in the form of a secretion effused on the surface of the serous membrane, must be self-evident. A special modification of structure or function of this membrane as necessary to their formation, would be a mere assumption, inasmuch as no change of either is frequently to be perceived ; and even in those cases in which a marked local change is present and has been known to precede the development of the disease in question, it cannot be regarded either as a necessary or essential condition of the existence of the latter. It may indeed be regarded as a law, that the speciality of a morbid product, of the nature of those of which I am now treating, is entirely independent of any local agency whatever. The trite but important remark, that hundreds and thousands of individuals are daily affected, for example, with inflammation, without this local disease being followed by any other than its usual effects, places in the clearest light the necessity of a previously existing modification of the economy, as the immediate and essential condition of the speciality of the heterologous formations when they occur in conjunction with inflammation. This may be illustrated by the following case, which is by no means rare : an individual has a tumour on the external surface of the body, presenting the characters of one or more of the varieties of scirrhus or cephaloma. He has an attack of pneumonia, pleurisy, or both, of which he dies in the course of a few days, one or two weeks. On examining the diseased lung or pleura, we find, instead of an effusion of serosity, coagulable lymph, or pus—the usual products of inflammation,—that the lung is converted into a solid mass resembling a section of fresh pork : it is in the state of scirrhus ; and the pleura is studded with tumours of various sizes composed of a similar kind of substance. It were vain to seek for an explanation of these appearances, by attributing them to the contamination of the blood from the absorption of the carcinomatous matter contained in the original tumour ; for admitting such to be the case, the difficulty would remain the same as regards the origin of the

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latter. Besides, similar cases occur, subsequently to inflammation, in individuals who have no such tumours in any other part of the body, and therefore we must look beyond the local cause for an explanation of the origin of the disease in both.

Formation of Carcinoma in the blood.—The presence of the heterologous substance which constitutes the several varieties of both species of carcinoma in the blood is a circumstance of great importance, and unless it be clearly demonstrated to arise in consequence of a modification of the blood itself,—in whatever manner produced,—we should find it impossible to explain many of the phenomena which it presents, more especially those which accompany its formation in the molecular structure of organs, and on the free surface of membranes. The following facts may be adduced as furnishing strong evidence that the formation of this substance takes place in the blood, whether it be found in this fluid alone, or in other parts of the body at the same time; 1st, the presence of this substance in the vessels which ramify in carcinomatous tumours, or in their immediate vicinity; 2d, in the vessels of a portion or of the whole of an organ, to the former of which this substance is exclusively confined, and can be traced from the trunks into the branches and capillaries; 3d, in vessels having no direct communication with an organ affected with the same disease, as, for example, when it is confined to a small extent of the vena portæ; and lastly, in blood which has been effused into the cellular tissue, and on the surface of organs.

The divisions of the vascular system in which the carcinomatous substance has been observed are the venous and capillary,—a circumstance which may be ascribed to the contractile power of the arteries preventing, under ordinary circumstances, the blood from accumulating, and, consequently, this substance from forming within them, and not to any peculiarity of function exercised by the former.

The appearances which the carcinomatous formation presents in the blood are very various, and sometimes perfectly similar to those which mark its presence in the substance or on the surface of organs. In large veins, such as the vena portæ and its branches, the emulgent vein, &c. it may present the lardaceous, mammary, medullary, or hæmatoid characters, all in the same venous trunk. These varieties of the carcinomatous formation may be found mixed together in minute quantities, or isolated into masses so conspicuous that we can readily distinguish them from one another; sometimes they lie merely in contact with the internal parietes of the vein; at other times they are united to the latter by means of a thin layer of colourless fibrine; or minute blood-vessels pass from the one into the other, and are often very numerous and remarkably conspicuous in the cerebriform matter.

The *formation* of these varieties of carcinoma in the blood cannot remain a matter of doubt to those who have had occasion to examine the appearances I have described. That the presence of an organized product in the blood can have no other origin than the blood itself, and that such a product cannot be introduced into this fluid by absorption or otherwise, are facts so obvious that I shall not attempt any farther illustration of them.

From this view of the origin of carcinoma, its formation in the intimate structure and on the free surface of organs, after the manner of nutrition and secretion, follows as a natural consequence. The material element of the disease is separated from the blood and deposited under a variety of circumstances which modify in a greater or less degree the form, bulk, colour, and consistence which it afterwards presents, in the several periods of its development. We cannot, therefore, limit the seat of this disease to any one tissue, or ascribe its origin to any modification of structure or special organization, as has been done by several pathologists. Perhaps the most ingenious essay that has been

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made to explain the local origin of carcinoma is that of Dr. Hodgkin, published in the fifteenth volume of the Medico-Chirurgical Transactions. Dr. Hodgkin has endeavoured to show that the presence of a serous membrane, having a cystiform arrangement, is necessary for the production of carcinoma and some other heterologous formations. The existence of the former precedes, he believes, the formation of the latter; and, consequently, is at once the seat and origin of the disease. That such is the manner in which carcinoma and several other heterologous products are sometimes formed I can have no doubt, inasmuch as I have seen them as described by Dr. Hodgkin. But there is here no new law in operation, nor even an exception established to that the principles of which I have explained. Cysts are a very simple modification of a serous membrane, they partake of the structure and functions of the latter, and consequently are subject to similar diseases. If, therefore, such cysts should exist in an individual having the cancerous *diathesis*, they may, in the same manner as a natural serous membrane, become the seat of any variety of carcinoma. But although carcinomatous tumours such as those described by Dr. Hodgkin are found contained in cysts, attached, single, or in groups, and covered by a reflected serous membrane, these tumours may, and frequently do, not originate in the cysts. They may form in the cellular tissue external to the cysts, and during their development project inwards, carrying before them as their common envelop the internal and serous lining of the latter. Such, in fact, is seen to be the origin of these tumours in most of the cysts represented by Dr. Hodgkin in the work referred to. They are situated external to the cyst, are supplied with vessels which do not belong to the cyst, and are placed in the same circumstances as tumours formed in the cellular tissue when no cyst is present.

As an objection to the general application of the cystic origin of tumours, it may be observed that the presence of cysts in the liver, walls of the stomach, lungs, kidneys, brain, lymphatic glands, spleen and blood, is not to be detected at any period of the development of carcinoma, and therefore, when they do occur in other organs, as the ovaries, testes, mammæ, &c. they must be regarded as a mere coincidence, or as a consequence of the disease, and not as a cause or necessary condition of it.

After what I have just said, it will not be expected that I shall do more than notice that vague and unphilosophical theory which maintained that the formation of carcinoma depended on the previous existence of an accidental organization, which received the name of *hydatid*: how far such was the appellation it should have received, must now be regarded as a matter of indifference.

Mr. Abernethy referred all adventitious formations to the coagulable part of the blood as their origin, and fixed their seat in the cellular tissue, in the parenchyma, and on the surface of organs. This plastic substance was supposed by him to be effused under one or other of these circumstances, to become organized, and to derive the materials of its growth from the vascular system of the surrounding parts: hence the term *fleshy* or *sarcomatous* given by him to these formations. It will readily be seen that this view of the seat and origin of adventitious formations is both imperfect and inaccurate. Many of these formations are not organized,—not *tissues*, as he believed, and as they were described to be about the same time by Laennec, but *amorphous* masses, all the changes which they undergo being dependent on the influence of external agents. Although the classification of these formations proposed by Mr. Abernethy was productive of some advantage, it is one which cannot now be employed, because of its assigning to them fictitious characters on the one hand, and, on the other, confounding under the same head diseases of an entirely opposite nature.

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The same observation may be applied to the classification proposed by Andral. He has brought together in the same class analogous and heterologous formations,—for example, accidental serous and cellular tissue, scirrhus and fungus hæmatodes, because, he says, they present one common and essential character, that of being organized. But even admitting that this latter circumstance justifies the bringing together adventitious formations so dissimilar in their nature and purposes, it is by no means founded in fact; for, as I have already stated, there are a certain number of the heterologous products which I have comprehended under carcinoma, that are *not* organized, and on which account I have made a distinct species of them. These have been included by Andral among the organized heterologous formations, contrary to the principles of his own classification.

With regard to the origin of these formations Andral refers them to a modification of secretion, consequently they may form wherever this function is accomplished; the speciality of each depending on a previous modification of the economy in general, or of the functions of nutrition, innervation, or hæmatosis in particular.

The only other opinion to which I shall at present allude regarding the seat and origin of these formations, considered in an anatomico-pathological point of view, is that of Cruveilhier. This pathologist regards all organic transformations and degenerations (as he calls them) as exclusively the result of the deposition of morbid products in the *cellular element* of organs. He believes that the *tissus propres* of organs are incapable of undergoing any organic lesion except hypertrophy and atrophy. Both these statements I have shewn to be discordant with facts. The cellular tissue is not the exclusive seat of these products, nor are the *tissus propres* of organs exempted from their presence, in proof of which I must refer the reader to what I have said of the mode of formation of carcinoma in the liver and stomach, where it can be distinctly seen to form in the molecular structure, or what I conceive to be the proper tissue of organs.

The source whence these products are derived is, according to this author, the venous capillary system, to which I formerly alluded, and assigned a reason for their being found in these vessels, and especially in the venous trunks and their larger branches.

The extent and importance of the present subject renders it necessary to devote another fasciculus to the description of the physical, chemical, anatomical, and physiological characters of carcinoma, together with the representations of the principal varieties of the disease which I have pointed out.

DESCRIPTION OF THE PLATES.

PLATE I.

Fig. 1 represents the stomach laid open in the direction of the small curvature from the cardia to the pylorus. A, œsophagus; B, duodenum; C, pylorus; D, cardia; E, fundus of stomach; FF, GG, muscular coat of the pylorus, and body of the stomach presenting the type of scirrhus of this organ. The muscular and cellular bands are distinguished from each other by the grey semi-transparent aspect of the former, and the dull white colour of the latter. HH, hypertrophy of the same coat at the cardia and in the œsophagus. K, lobulated tumours formed in the submucous and mucous coats of the pyloric portion of the stomach, and at the cardia. These tumours presented the mammary characters, which are better marked in the following figures.

Fig. 2. The pyloric portion of another stomach laid open and spread out. A, pylorus; B, duodenum; C, body of stomach. DD, muscular, EE, submucous, FF, mucous coats. The muscular coat presents the same general arrangement as in the preceding figure, but is much less distinct in some places, has a yellowish tinge, and is less transparent. The mucous and submucous coats are very conspicuous, and are affected nearly to the same degree. Both of them present the lardaceous and mammary aspect. The development of the disease in the mucous and submucous tissues is carried to an extraordinary extent, forming a broad, elevated band, HH, surrounding the whole circumference of the stomach; and tumours, one of which, G, is nearly as large as a goose's egg.

Fig. 3 is a section of this tumour. A, duodenum; B, pylorus; C, muscular coat; D, submucous coat forming the greater part of the tumour, and E, the mucous coat, which is seen to vary much in thickness in different parts of its extent. In these two figures in particular, we have not only fine specimens of carcinoma affecting the three coats of the stomach, but also three forms of the disease; viz. the scirrhus, lardaceous, and mammary. The last is well marked in the submucous tissue, D, of *fig. 3*. There was also a tendency to the cerebriform and hæmatoid at several points of the surface of the tumours, marked by circumscribed effusions of blood.

PLATE II.

Fig. 1 illustrates very beautifully the transition of scirrhus to mammary sarcoma, of the mammary to the medullary, and of this last to the hæmatoid. A, pylorus; B, muscular coat, presenting faintly but distinctly the characters of scirrhus; C, submucous coat presenting those of mammary sarcoma; and D the mucous membrane, pulpy, brain-like, vascular, and bloody. The mucous membrane of the whole of the pyloric portion of the stomach was red, vascular, and projecting more or less above the general surface of the stomach, as at E. It contained here and there little round masses of the brain-like matter, seen also at F.

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Fig. 2 represents a large, flat, lobulated tumour, B, situated near the cardiac orifice of the stomach. It was formed by all the coats of this organ, but principally by the mucous and submucous, which were converted into the mammary and medullary variety of carcinoma, the muscular coat still presenting the scirrhus character. Some parts of it were quite pulpy, particularly at its centre, C; and on various points of its surface hemorrhage had taken place. When cut and pressed, great quantities of the cerebriform matter flowed out, tinged with blood; and when this substance was nearly all removed, there remained behind a loose, spongy, cellular tissue, plentifully supplied with minute bloodvessels. In *fig. 3* we have a still more remarkable specimen of carcinoma, in which the scirrhus, lardaceous, mammary, medullary, fungoid, and hæmatoid forms of the disease were all combined. The stomach is represented laid open from the cardia, A, to near the pylorus B. The disease is situated around the cardia, and small curvature of the stomach; forms tumours, CCC, of various shapes and sizes, slightly tinged with bile; some of them are entire, others present a slight abrasion of their surface, DD, which is vascular and bloody; others are broken down into masses of greater or less bulk, EE, and formed of fluid and coagulated blood and cerebriform matter. Some of them were entirely composed of this matter, others nearly pale, more or less red, and vascular. The lymphatic glands, G, in the neighbourhood of the stomach, were enlarged, some of them firm, others, H, soft, pulpy, and vascular.

PLATE III.

Fig. 1 represents carcinoma of the rectum. BB, the muscular, and CC, the submucous coats of the intestine. They are nearly equally increased in thickness; both of them have a somewhat fibriform arrangement, but the submucous and intermuscular cellular tissue has a dull yellow lardaceous aspect, an appearance very common in cancer of the walls of the intestines. The mucous membrane, DDD, is in some parts destroyed, and of a dull yellowish-green colour; in others, thick, elevated into irregular masses, soft or pulpy, vascular and bloody. The lymphatic glands, EE, were very hard, and composed of a grey semitransparent substance; and a number of round bodies, F, of a similar nature, occupied the peritoneal surface of the intestine. *Fig. 2* has been given to shew the appearance of a cancerous ulcer of the stomach. D, the bottom of the ulcer, of a dull yellow and greenish tint, formed by the submucous tissue, and surrounded by a broad, rounded, elevated border, E, consisting of the mucous membrane and the former tissue, the thickness of both of which is considerably increased. The plicæ, F, of the mucous membrane, around the ulcer, are also seen to be much enlarged. B, the œsophagus; C, the cardiac orifice. A vertical section of the ulcer is shewn at *fig. 3*; A, the bottom of the ulcer; B, the vascular surface of the mucous membrane; C, a section of it, shewing its thickness; D, the thickened and lardaceous submucous tissue, terminating gradually in the healthy coats of the stomach, beyond the limits of the ulcer. The mucous coat, C, is composed chiefly of the mammary substance, is firmest where it is in contact with the submucous coat, and becomes quite pulpy and brainlike towards its free surface, on which are seen little round eminences composed of a similar matter. The muscular coat, EE, is healthy. *Fig. 4* is a remarkably fine specimen of carcinoma confined to the mucous coat of a portion of the duodenum, with the exception of thickening of the muscular coat, A. The mucous membrane, BB, is in some places the eighth part of an inch in thickness, and raised into a number of convolutions of a

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brainlike aspect tinged here and there with red; the commencement of the hemorrhagic or hæmatoid state. The commencement of this disease in the mucous membrane is particularly well seen at DD, in the form of pale elongated elevations, entirely limited to the mucous tissue. In many parts the villousities, EE, are very large and prominent, quite pale, and were apparently composed of cerebriform matter. A great number of lacteals, GGG, arise from the mucous membrane; some of them, as at F, appear to proceed from the enlarged villousities. They were filled with a white creamy fluid, and are seen passing into the mesenteric glands, which are greatly enlarged and composed of vascular cerebriform matter. Another example of carcinoma of the mucous membrane, that in which it assumes the form of what is called a cauliflower excrescence, has been represented in the female bladder, *fig. 5*. A, the urethra; BB, the divided walls of the bladder. C, a multitude of globular and pyriform bodies; varying from the size of a pin's head to that of hemp-seed; some of them attached by a narrow pedicle, others by a broad basis. They are of a pale bluish-grey colour, felt rather soft, and, when divided, a somewhat milky fluid could easily be pressed out of the largest of them. They were formed in the mucous tissue. Near the orifice of the urethra are situated three pediculated tumours, D, two of the size of a common mulberry, and having the external configuration of that fruit. They are of a rose red colour, were composed of a cerebriform substance contained in a fine filamentous tissue, and in which a multitude of minute bloodvessels ramified. These terminated in several small trunks, which passed along the pedicle of each tumour, and communicated with the vascular system of the submucous tissue. E, perforation of the bladder from cancer of the uterus.

PLATE IV.

This plate represents carcinoma of the liver in the scirrhus, lardaceous, mammary, medullary, and hæmatoid forms; the manner in which the disease is developed in the acini of the liver, and its presence in the blood. In *fig. 1* it appears in the form of round tumours, AA, varying from the size of a pea to that of a small orange, single or aggregated. These tumours are composed chiefly of a lardaceous or mammary substance, with a greyish-coloured central depression; some of them have a uniform aspect, others a radiated fibriform arrangement, and contained a considerable quantity of cerebriform matter, which could be squeezed out from the cut surface. Minute vessels are seen ramifying in most of them from their circumference towards their centre; and in one of them, C, the central portion of which was composed of cerebriform matter, the vascularity is very conspicuous. The cerebriform characters of these tumours, as well as the hæmatoid, are well marked at B, the whole of the group in this situation being composed of this substance, a tissue of minute vessels, and a greater or less quantity of effused blood. The external characters of these tumours are represented at DD, on the surface of the liver. They are marked by a central depression, a radiated structure, and distribution of the bloodvessels. In *fig. 2* is illustrated the molecular formation of the disease in the acini. The heterologous deposit is seen at AA, making its appearance in two, three, or more, contiguous acini, which are distinguished from the others by their being pale or nearly white. They form small tumours of the size of a hemp-seed or pea, in which the separation of the acini is quite distinct. In other points of the substance of the liver, as at BB, they have acquired double or treble the bulk of the former by the extension of the deposit to a greater number of acini, which are still visible throughout

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the whole of their substance ; in some of them, the bulk of which is very considerable, the form and arrangement of these bodies can be readily recognized. At D, and more especially E, where the isolated tumours are grouped together into large masses, the primitive structure, or that just described, has disappeared, and the substance of the liver is converted into a grey, milky, yellow, rose-coloured substance, presenting here and there the lardaceous and mammary aspect and consistence, and, when pressed, discharging minute drops of a milky-looking fluid. Bloodvessels were seen ramifying in most of them, and hemorrhage had taken place in different points of their substance, as represented at FF and GG, in which latter points are collected small round or irregular masses of fibrine. *Fig. 3* represents a tumour, A, removed from the substance of the liver, with a number of small veins, BB, passing out from it. These veins terminated in the substance of the tumour, and contained a brain-like matter as well as the tumour itself. This matter was in greatest quantity in the veins where they were in contact with the tumour, as may be seen in the figure, by their size at this part. Two of them, CC, are seen communicating with a large trunk, D, into which the cerebriform matter could be made to pass by pressing on the former. *Fig. 4* is another example of the carcinomatous formation in veins. A, is a section of a large tumour from the liver ; B, one of the divisions of a large branch of the vena portæ, H, ramifying in the carcinomatous substance which is shewn exposed by a lateral section. At C, the vein is seen passing out of the tumour, and connected with a smaller branch, D, situated on the surface of the latter. Into this branch, the walls of which are marked at E, three very small branches terminate, after passing out of the substance of the tumour. From these and the large branches, B and D, the carcinomatous formation passes into the trunk, H, and is divided throughout its whole length, G, to shew that it was similar to the substance of the tumour. It was distinctly vascular, adhered to the surface of the vein, and terminated at H, in a rounded extremity, covered by a smooth transparent membrane. *Fig. 5* represents that state of the veins in carcinoma of the liver which gives rise to ascites. A, is a branch of the vena portæ, of considerable size, laid open ; BB, cancerous tumours, projecting into it, C C, without perforating it ; and thus occasioning its entire obliteration as seen at D. The presence of the carcinomatous matter within the veins, as shown in the former figure, is another mode of obliteration, or obstruction to the circulation in the liver, and consequently a cause of ascites ; and a third mode is that which is effected by compression, the opposite parietes of the veins being brought into contact and becoming united under the pressure of the tumours through which they pass. Veins obliterated in this manner are represented at EE, *fig. 1*.

Plate 1.

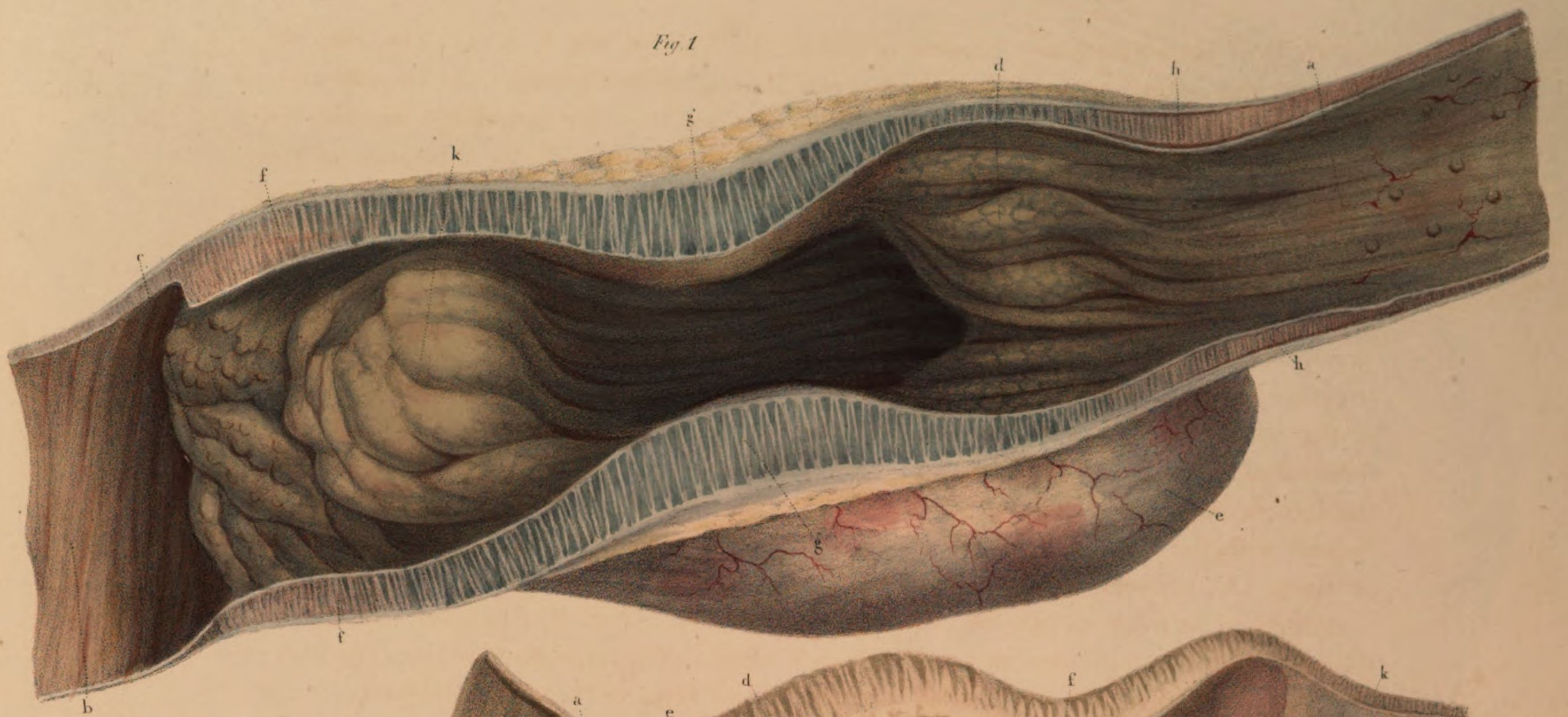


Fig. 2.



Fig. 3.



Plate II..

Fig. 3.

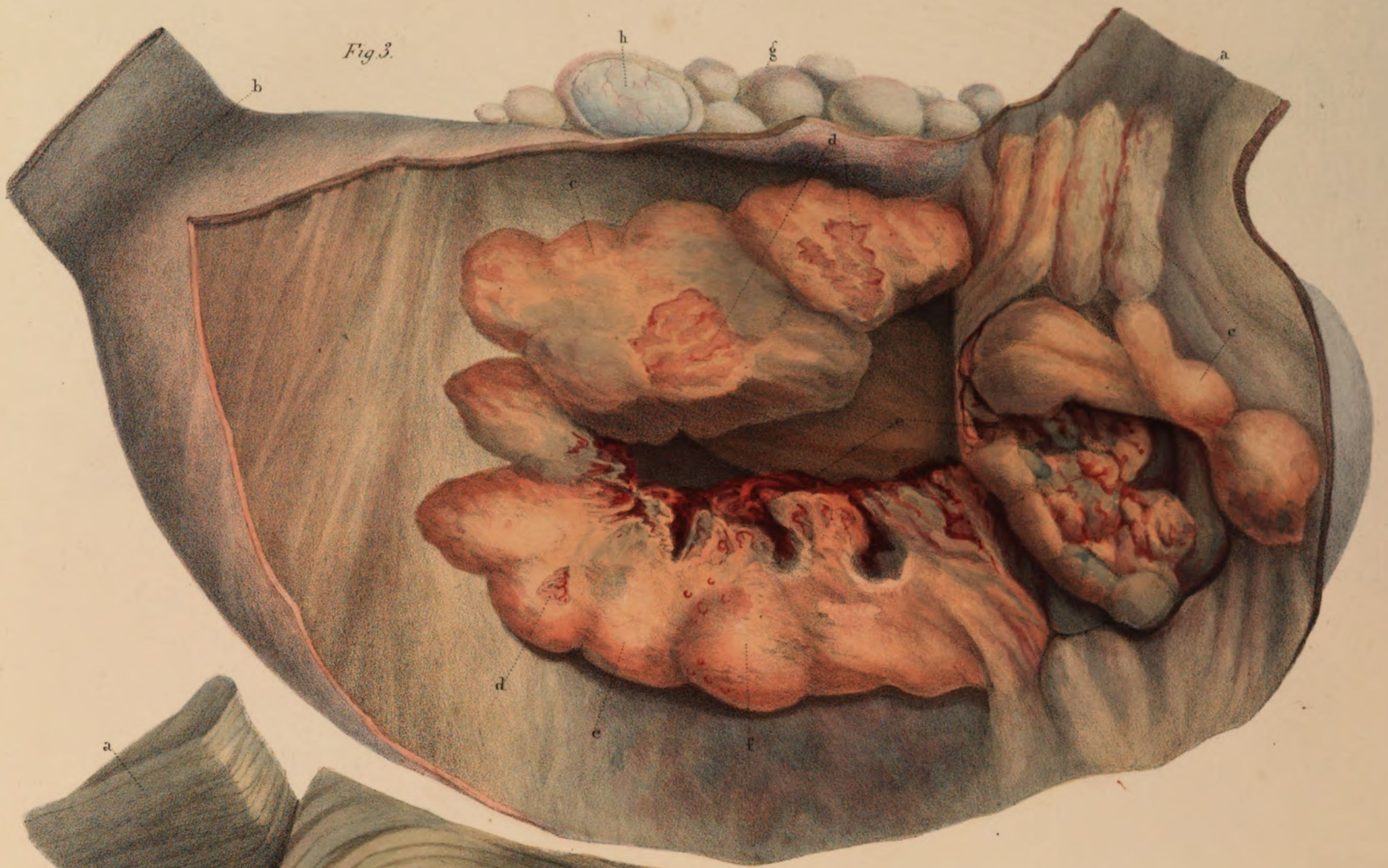


Fig. 2.



Fig. 1.

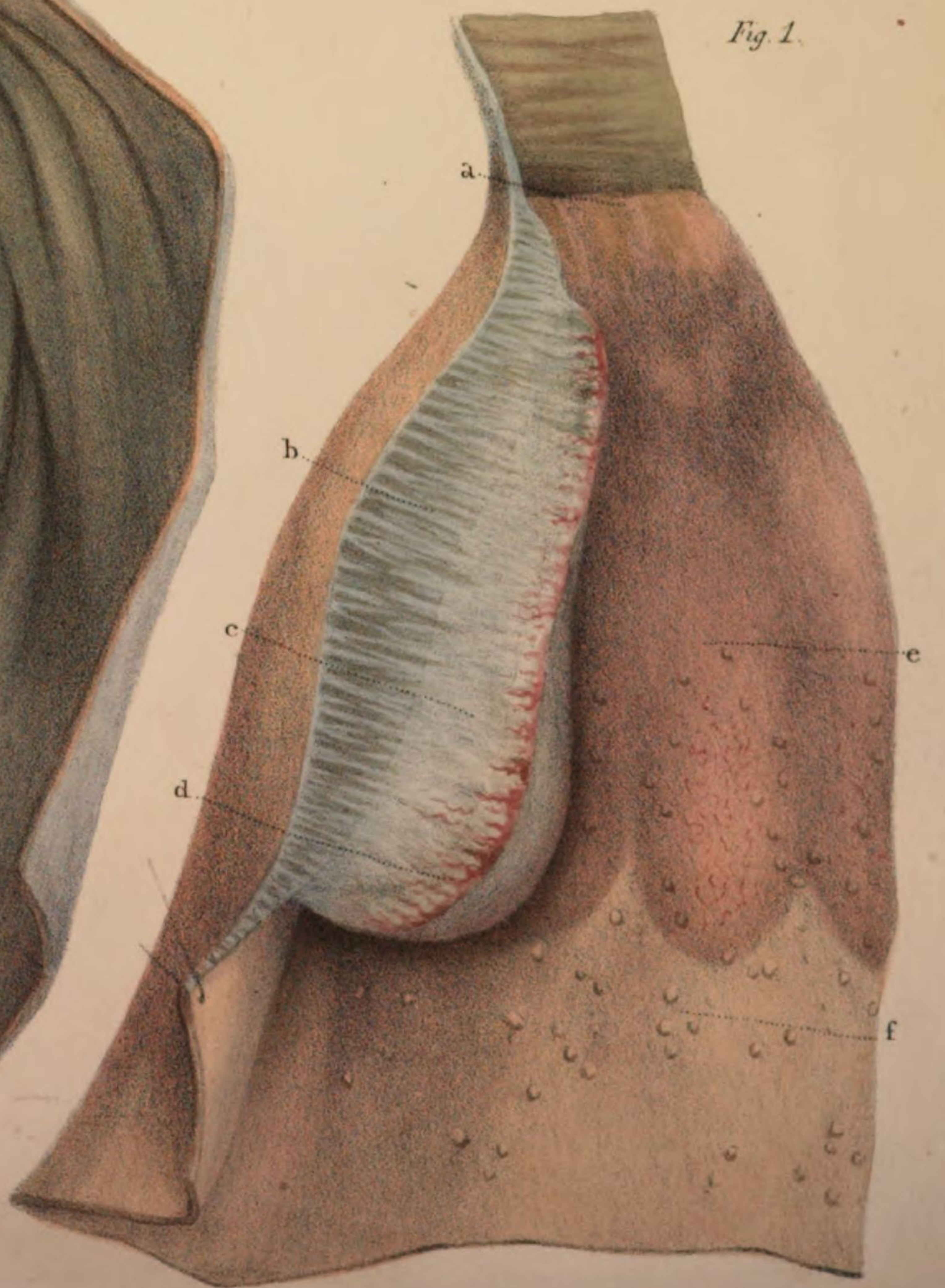


Plate III.

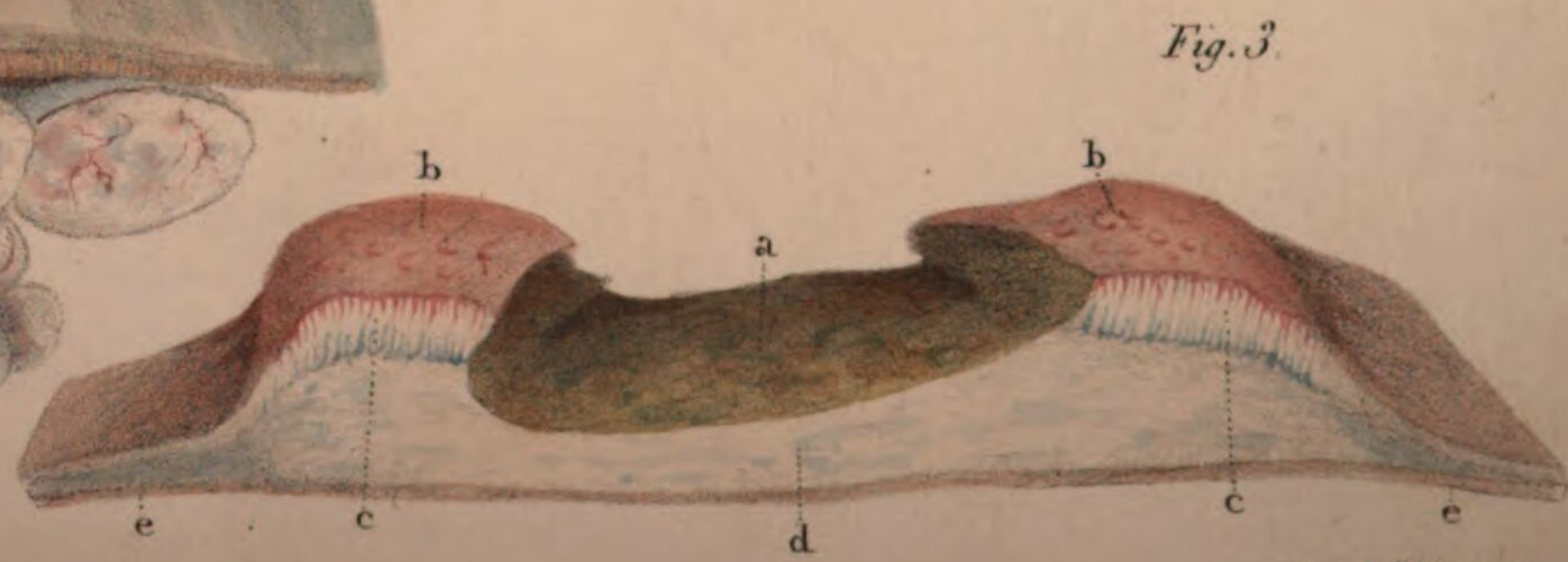
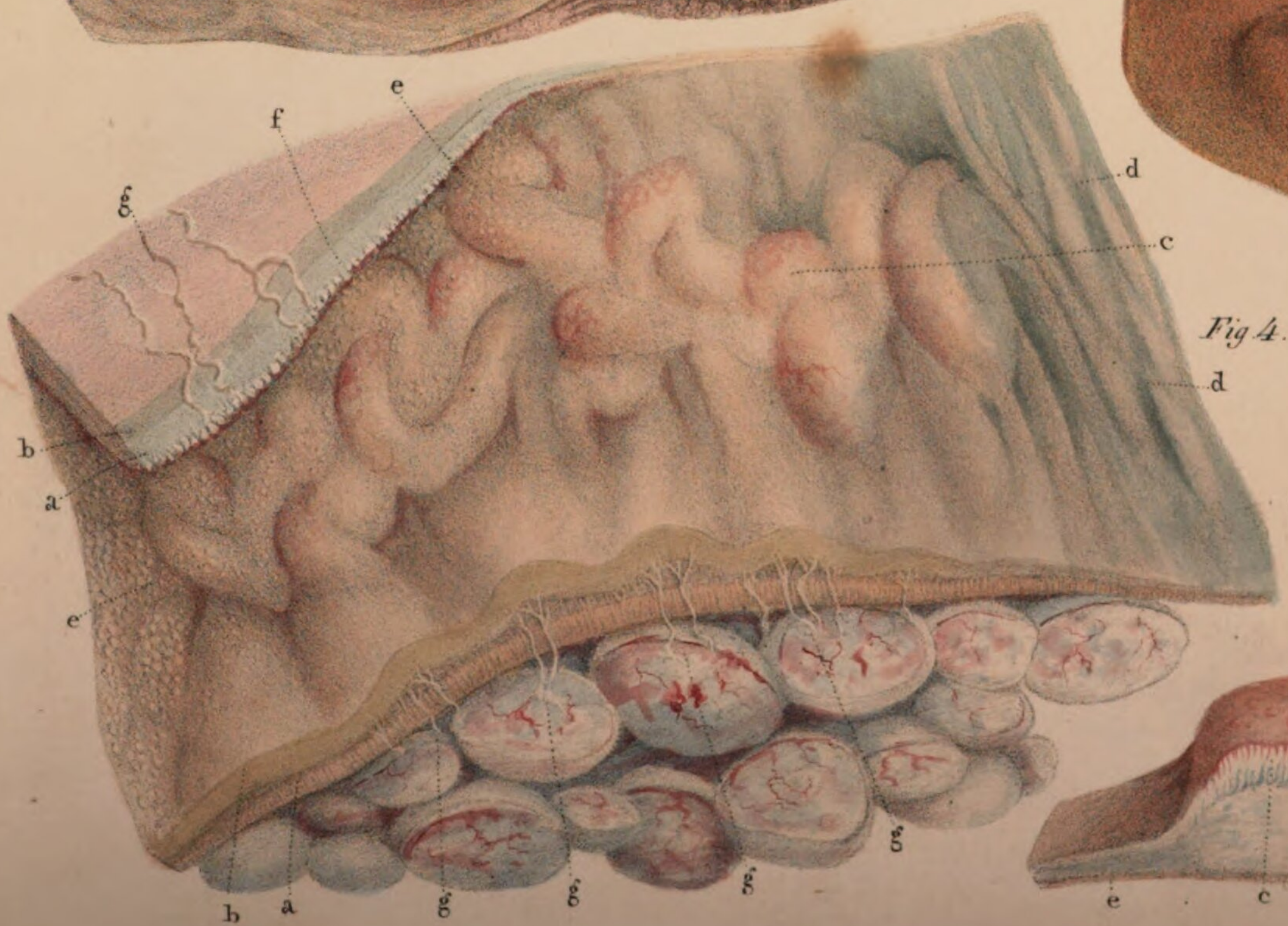
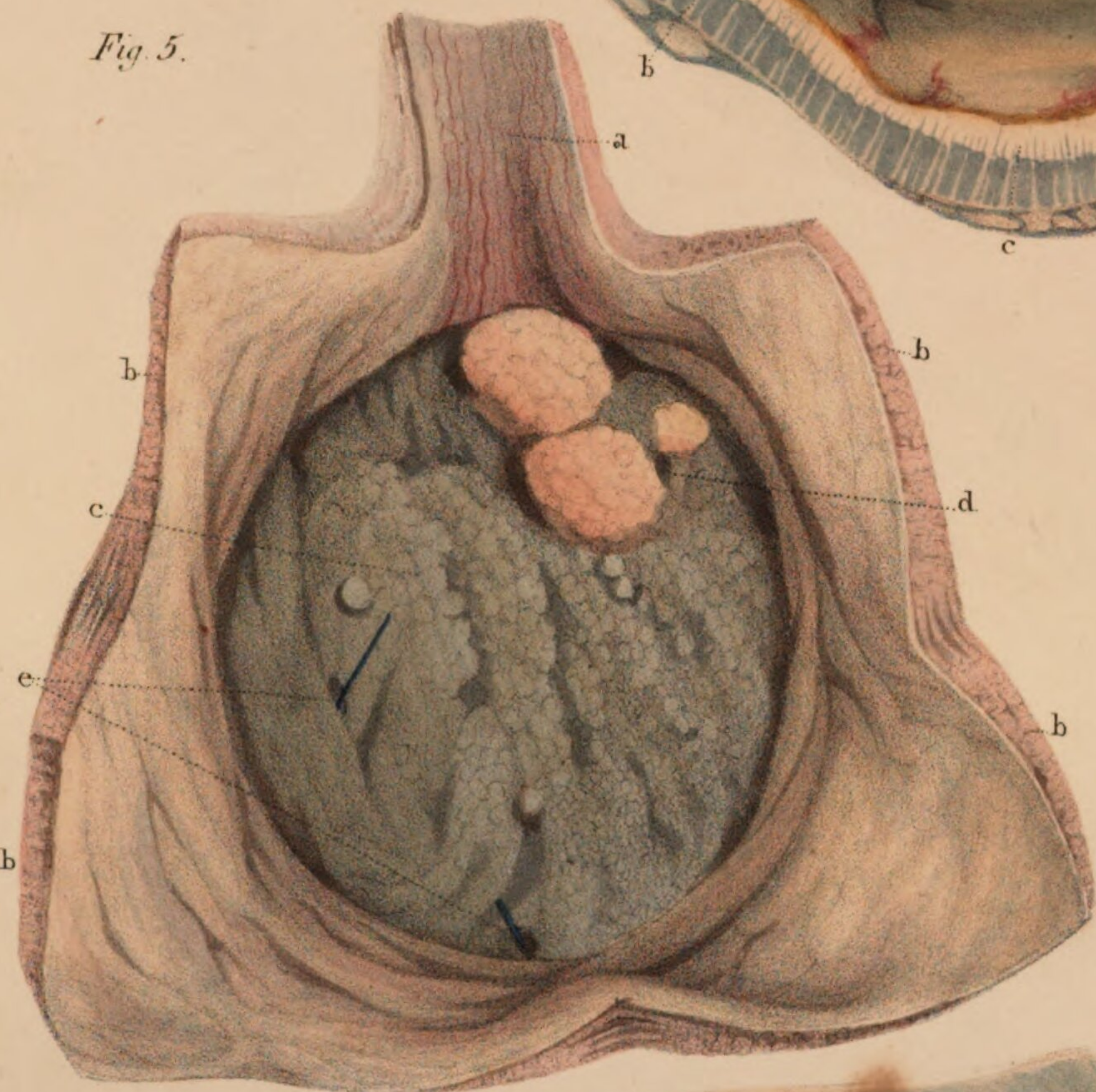


Plate IV.

Fig. 1.



Fig. 5.

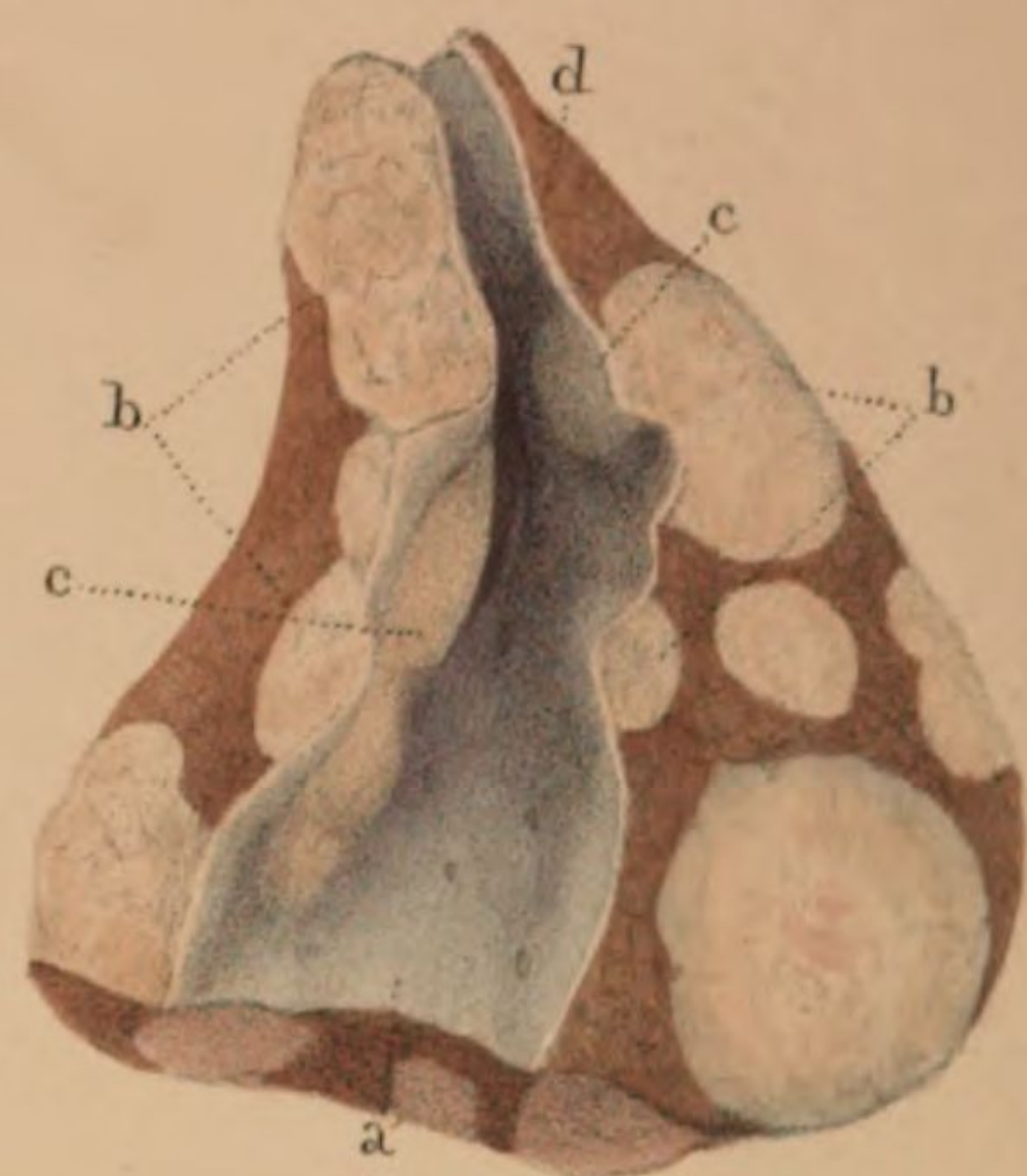


Fig. 3.

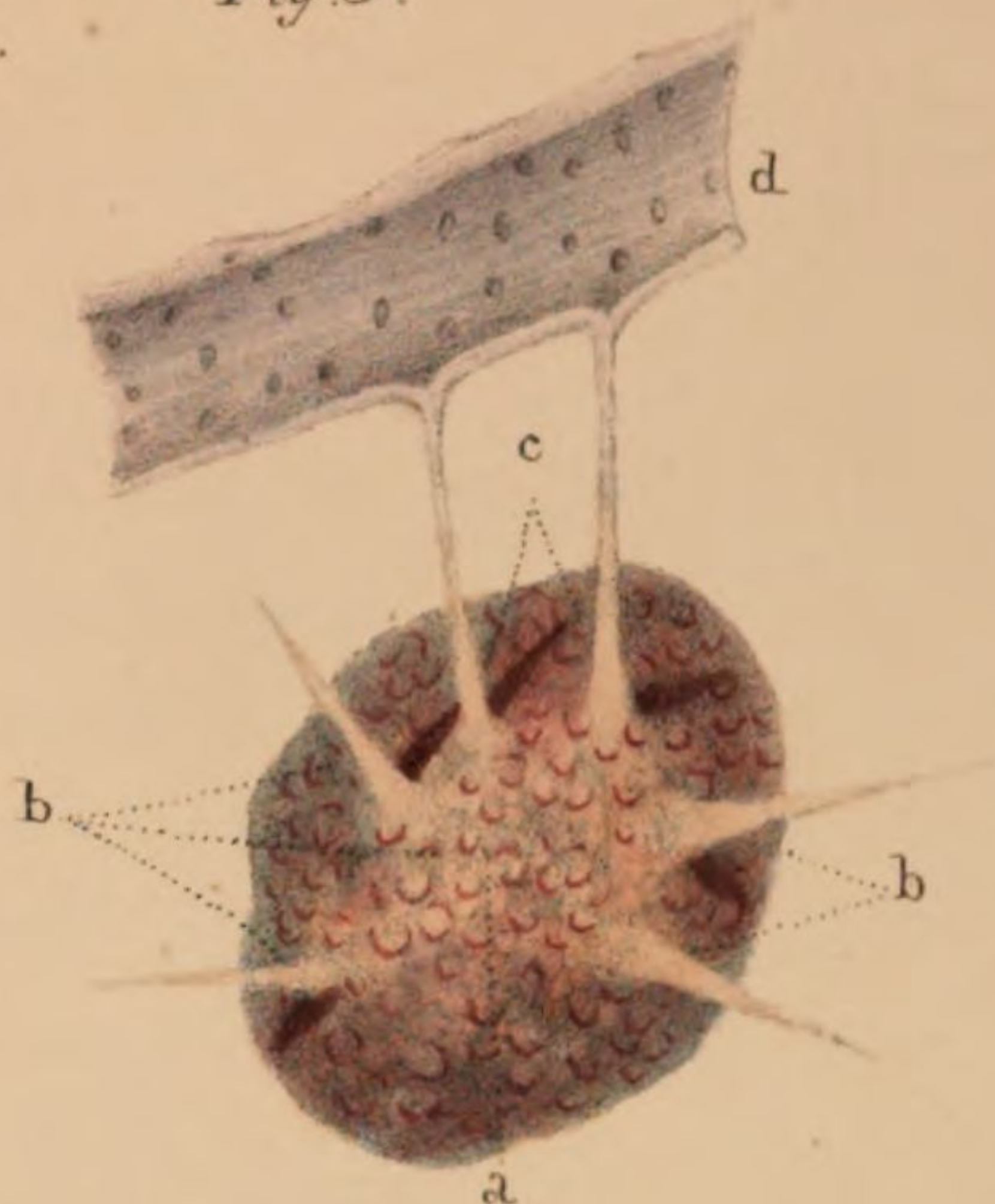


Fig. 2.



Fig. 4.



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Physical Characters of Carcinoma.—The physical characters of this disease comprehend the form, bulk, colour, and consistence which it presents in the different tissues and organs of the body, and in the several periods of its development.

Form.—Carcinoma presents considerable variety of form. In its first stage, and when the material of which it is composed is deposited after the manner of nutrition, Carcinoma assumes the particular form or structure of the organ which it affects, as that of the liver and stomach, the acini of the former, and the muscular, cellular, and mucous tissues of the latter, determining, in these organs respectively, the primary form of the disease. In the brain, lymphatic glands, and testes, we cannot, however, perceive any particular arrangement of the carcinomatous matter at this early stage, either on account of the colour, homogeneous aspect, or minute structure of these organs preventing us from detecting its presence and the manner in which it is deposited.

At a more advanced stage, the forms which the carcinomatous matter derives from the structure of the parts in which it is deposited disappear, and those which it afterwards presents are determined in great measure by external circumstances. The most important of these forms are the *Tuberiform*, *Stratiform*, and *Ramiform*.

1. *Tuberiform Arrangement of the Carcinomatous Matter.*—This form of the carcinomatous matter is by far the most frequent, and presents considerable variety. When this matter is deposited in organs possessing a uniform density, and in parts submitted on all sides to an equal degree of pressure, it assumes a globular form. On natural and accidental serous surfaces, although at first globular, it frequently becomes pyriform, either on account of the mode of its attachment, or of less resistance being opposed to its growth in one direction than in another. It assumes a fungiform shape when placed in circumstances which facilitate its lateral or retard its anterior development, as when it meets with a dense unyielding substance during its progress, or, having pierced the skin, is subjected to pressure. When accumulated in separate portions of the cellular tissue into rounded masses, grouped together, and included within a common capsule, it generally presents a lobulated appearance; and in the submucous tissue in particular, it frequently exhibits the external arrangement of the cauliflower or mulberry. That appearance of Carcinoma which resembles the structure of the pancreas, depends generally on the agglomeration of very small globular or pyriform tumours, separated from one another by cellular or cellulo-fibrous tissue, but inclosed in a common capsule.

2. *Stratiform Arrangement of the Carcinomatous Matter.*—The carcinomatous matter having this form is chiefly met with in the subserous cellular tissue. Although it may be deposited in layers of various extent which present no definite arrangement, it more frequently assumes the form of thin circular patches, varying from the breadth of a pin's head to an inch or more in diameter, and presenting an appearance similar to what might be imagined to follow the infusion of a small quantity of milk into a number of isolated points of the subserous cellular tissue. Patches of this kind, which are composed of a substance having the colour and consistence of cream or milk, are most frequently met with beneath the pia mater and pleura pulmonalis, and are remarkably conspicuous in the latter situation on account of their white pearly aspect contrasting so strongly with the surrounding dark colour of the lungs. These patches may occur in the situations I have named without the substance of the brain or lungs presenting any trace of Carcinoma; but I have never met with them unless when the disease existed in some other organ, as the breast, eye, liver, stomach, kidney, or uterus. In some cases, lymphatics filled with fluid carcinomatous matter are observed to communicate with the patches; in other cases no such vessels are observed.

[*Carcinoma*, 3.]

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3. *Ramiform Arrangement of the Carcinomatous Matter.*—The ramiform arrangement of the carcinomatous matter depends, as has been already stated, on the presence of this matter in the veins. I have represented it in *figs. 3 and 4, Plate IV.* of the second fasciculus, occupying the veins situated in carcinomatous tumours of the liver, and passing beyond the latter into larger branches. When such tumours are divided and submitted to pressure, we can often perceive the carcinomatous matter issuing from a number of small circular orifices in the form of a creamy fluid, and if these orifices are attentively examined by a careful dissection of the tumour from its cut surface towards its circumference, we find that they are the cut extremities of veins filled with this matter to a greater or less extent beyond the tumour. There is no organ in which this arrangement of the carcinomatous matter is so conspicuously seen as the kidney. The whole of the venous system of this organ, including the emulgent vein to its termination in the vena cava, is sometimes found completely distended with this matter, either in a fluid state, of the consistence of brain, or as firm as the pancreas. When a kidney thus affected is divided, it appears as if it were formed of a multitude of encysted tumours of various sizes, on account of the carcinomatous matter being contained within, and bounded by, the walls of the cut extremities of the veins. This form of Carcinoma of the kidney is easily ascertained by dissection, or by the introduction of a probe from the emulgent vein into its branches.

A similar arrangement is sometimes remarkably conspicuous in Carcinoma of the stomach. Not only are the minute veins which ramify beneath the mucous membrane in the vicinity of the disease, filled with the carcinomatous matter, but also the larger branches seen on the external surface of the stomach, and the coronary veins in which they terminate. The abdominal division of the vena portæ furnishes us with a remarkable example of the ramiform arrangement of Carcinoma, isolated from any organ affected with this disease.

There is another variety of form of the carcinomatous matter which may be noticed in this place, as it may be regarded as a modification of the preceding. It is that which is observed when this matter is contained in the lymphatics and lacteals, and which is derived from the particular form and distribution of these vessels. The lacteals more frequently, perhaps, than the lymphatics contain this matter, and are sometimes seen in great numbers quite filled and even distended with it, on the surface of the stomach and intestines, and between the folds of the mesentery, in Carcinoma of these organs. It is not correct to say that the lymphatic glands situated in the neighbourhood of an organ, as the mamma, affected with Carcinoma, always become diseased through the medium of absorption; for we often find these glands extensively diseased while the lymphatics present no lesion whatever, that is to say, they may be perfectly pale and transparent, and may not contain the slightest trace of the carcinomatous matter. Such are the principal forms of Carcinoma, whether we consider the disease in a general or special point of view. They are certainly not equally prevalent, nor precisely the same in both species, *viz.* in Scirrhus and Cephaloma, nor in the several varieties of each; but they are found to occur in all the varieties, and as the modifications which they present, in this respect, are very unimportant, it is not necessary to notice them more particularly.

Bulk.—The quantity of the carcinomatous matter deposited in the molecular structure or on the free surface of organs is extremely various, but it is perhaps never so great in the former as on the latter. In the liver it may vary from the size of a pin's head to that of an orange. In softer or more yielding organs, as the lungs, testis, and even the mamma, it may equal in bulk the head of an infant or of an adult; and in the inter-

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muscular and subcutaneous cellular tissue, its bulk is sometimes still more considerable. I am now speaking of individual tumours, and not of those masses formed by the aggregation of tumours during their progressive development, as occurs in the liver, lungs, &c. nor of those produced in a similar manner in the abdominal cavity posterior to the peritoneum, in Carcinoma of the mesenteric glands.

The influence of pressure in favouring or retarding the development of carcinomatous tumours, and consequently in modifying their bulk, is most conspicuously seen when they are situated near the external surface of the body. So long as their progress outwards is obstructed by an unyielding fibrous membrane, they often remain for a considerable time nearly stationary; but so soon as this obstacle is removed, they acquire a rapid increase of bulk. This rapid increase of bulk on the removal of all pressure is still more remarkable when these tumours project through the ulcerated integuments in the frightful form of bleeding fungi. But the best illustration of the influence of pressure on the development of carcinomatous tumours is met with in Carcinoma of the eye. A tumour which may have required several months before it reached the external surface of this organ, will, after it has been removed together with the whole contents of the orbit, reappear, and in one or two weeks acquire a much greater bulk than before the operation.

Independently, however, of this physical circumstance which modifies so conspicuously the bulk of these tumours, there is another of an opposite nature which requires to be particularly noticed on account of its constituting the distinctive character of the second species of Carcinoma. I allude to the physiological properties of the cephalomatous tumours, by means of which they possess within themselves the power of increasing their development to an almost unlimited extent. It is to the vascular organization, which I shall afterwards describe, of the tumours of this species that the rapidity of their growth and the great bulk which they attain are to be attributed, and which renders them less subject to the influence of pressure than those of the species Scirrhus. However, unless we were aware of the modifying influence of pressure, we should often be unable to explain why tumours possessing the same characters are subject to differences both as regards the rapidity of their development and the bulk which they acquire.

Colour.—The colour of Carcinoma differs greatly from that of any of the other Heterologous Formations. It is, therefore, a character of considerable importance, inasmuch as it frequently enables us to distinguish this disease from others of the same class; and we have already seen that it is chiefly by the same means that we are frequently led to a knowledge of the seat and forms of Carcinoma in the early stages of its development. It is most frequently white, with a shade of grey or blue; sometimes it inclines to yellow, brown, or red, in consequence of the colour of the organ affected with the disease, of the presence of blood, bile, pus, or other fluids in various proportions, or of some other accidental circumstance. But the principal modifications of colour of Carcinoma are seen in the several varieties of both species of the disease; these varieties, as I have already stated, resembling more or less in colour that of the organ or tissue whence have been derived their respective appellations, as that of cartilage, of the pancreas, of fresh or boiled pork, of coagulated albumen or fibrine, of the mammary glands, of the cerebral substance, or a mixture of the latter and blood.

Consistence of Carcinoma.—To none of the physical characters of Carcinoma has so much importance been attached as to that of consistence, but more especially to an increased degree of this property when considered in relation either to the disease itself

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or the tissues of the affected organ. Hence *Scirrhus*, a term which implies a state of induration, in consequence of its being frequently not only one of the first, but likewise one of the most marked changes which we are capable of perceiving in the affected organ, has been employed to characterize the early or occult stage of Carcinoma. The opposite condition of Carcinoma, that in which this disease presents a degree of consistence less than that of the organ which it affects, has been considered as a change succeeding to the state of induration or scirrhus, and the result of a process of softening, consequently, as indicating a more advanced period of the disease. But the degree of consistence of the Carcinomatous Formations is not an invariable character of a particular stage of their development; for these formations may, when first perceivable, be as hard as cartilage, soft as brain, or fluid as cream; or they may become soft or fluid after having remained for a greater or less length of time in a state of hardness.

This variety in the consistence of the Carcinomatous Formations depends on the following circumstances:—1st, The nature of the organ in which the carcinomatous deposit is contained: 2d, The elementary composition of the deposit: and, 3d, The subsequent changes occurring either in the deposit itself or in the tissues with which it is in contact.

1st. The structure, situation, connexion, and greater or less density of organs and tissues greatly modify the consistence of the carcinomatous deposit, either in consequence of a difference between the quantity of this substance relative to that of the tissues in which it is contained, or of a difference in the degree of resistance opposed by the latter to its accumulation or development. Thus it is more consistent in the liver than in the lungs or brain; in the skin than in the cellular tissue or a mucous membrane; in a tumour situated beneath a dense covering than on a free surface.

2d. Modifications in the composition of the carcinomatous deposit exercise a considerable influence over the degree of consistence which it presents, for we often meet with it possessing various degrees of consistence, when examined at the same stage of its development, and in the same or in different organs. Examples of this, although common in almost every organ of the body, are best seen where the carcinomatous deposit is collected into isolated masses containing little or none of the natural tissues, and where, consequently, its consistence must depend on the nature of the elements of which it is composed. It is found in this state in the cellular tissue and on serous surfaces; and more especially on accidental surfaces, such as those of sores formed by the destruction of the protruded portion of tumours, or after the removal of the breast, eye, testis, or other external parts affected with Carcinoma. In all these situations this substance may, at the same stage of its formation, present the opposite extremes of consistence, being in one case as hard as cartilage and more or less transparent, and in another as soft as brain, or quite fluid and opaque. These opposite extremes of consistence are most strikingly manifested in those fungiform tumours which arise from the bottom of the orbit after the extirpation of the eye, or from the cicatrix of the integuments after the removal of the breast in consequence of Carcinoma. In these two instances it is obvious that the stage of development of the tumours is the same in both; and that the difference of consistence which they present is no evidence of their being different in their nature is equally obvious, inasmuch as the hardest of them often assume, after a certain length of time, the consistence of the softest, a part or the whole of the dense transparent substance of which they are composed being gradually transformed into a soft brain-like pulpy mass. This process of transformation is also most conspicuous in those tumours which constitute the *vascular* or *organized*

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sarcoma of Mr. Abernethy, or in the classification which I have proposed, a variety of *Cephaloma*. Such tumours which at first are more or less transparent, presenting the appearance of a solid mass of firm albumen, coagulable lymph, or fibrin, become gradually opaque, soft, and pulpy, resembling foetal brain, and are then not to be distinguished from those carcinomatous tumours which from their commencement possess the cerebriform character. These examples will suffice to show that the carcinomatous deposit, besides being modified in its consistence by the tissues in which it is contained, is equally so in consequence of a difference in its composition; that its consistence may or may not be the same when first formed; that it may be either hard or soft at this period; and, consequently, that the latter state is not necessarily preceded by the former, as was maintained by Laennec and the greater number of pathologists who have published on this subject since his time.

3d. The last modification of consistence of the Carcinomatous Formations is that to which the attention of pathologists has almost exclusively been directed. It depends on a series of changes taking place either in the carcinomatous matter itself, the tissues with which it is in contact, or in both at the same time. But in order that these changes may be better understood, I shall first describe the chemical and anatomical characters of Carcinoma.

Chemical Characters of Carcinoma.—In order to ascertain the chemical composition of the several varieties of Scirrhomata and Cephalomata, it would be necessary to procure a sufficient quantity of the carcinomatous matter isolated from the tissues with which it is so frequently more or less intimately united or combined. The great difficulty, and, in many cases, the impossibility of obtaining it, in several of these varieties, in a separate state, has prevented the pathologist from determining accurately its chemical composition. Indeed the results of the analyses that have been published may be regarded as indicating the chemical composition of particular organs or tissues affected with Carcinoma, rather than of the carcinomatous matter itself. The most recent analysis of Carcinoma in the Scirrhomatous and Cephalomatous states is that published by Lobstein in his “*Traité d’Anatomie Pathologique*.” Seventy-two grains of scirrhus breast were found to contain—

Albumen	2 grains.
Gelatine	20 „
Fibrine	20 „
Fluid fatty matter	10 „
Water	20 „
	—
	72
	—

Seventy grains of the uterus in a state of scirrhus contained—

Gelatine	15 grains.
Fibrine	10 „
Fatty matter	10 „
Water	35 „
	—
	70
	—

According to the same author it would appear that the chemical composition of *Cephaloma* is not the same at different periods of its development: thus, in the first stage of the disease, or that of *crudity* as it is called, it was found to contain a greater

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proportion of gelatine than albumen; and in the second stage, or that of softening, that is to say, when the carcinomatous matter is of the consistence of soft brain, the albumen is in much greater quantity than the gelatine.

For the reasons already stated, it must be obvious that no great importance can be attached to these results of the chemical analysis of the carcinomatous matter. And, independent of the difference of composition which it must present from its admixture, in various proportions, with the same or different tissues, it is highly probable that it is likewise modified in this respect by the physiological influence of the organ in which it is formed, and the constitution of the individual in whom it exists.

Anatomical Characters of Carcinoma.—The most important circumstances illustrative of the anatomical characters of Carcinomatous Formations have already been pointed out, when treating of the specific divisions of the disease, its varieties of form, bulk and consistence, its seat and mode of formation. I shall, therefore, now examine more especially the structure or anatomical arrangement of the carcinomatous matter itself.

I formerly stated that the carcinomatous matter may exist in two states; that in the first state it has little or no tendency to become organized, its form and arrangement being determined chiefly by external circumstances; and that in the second it exhibits a greater or less tendency to become organized, possessing in itself properties by means of which its form, arrangement, and development are effected. The carcinomatous matter may, as we have seen, exist in three situations, viz. in the molecular structure of organs, on free surfaces, and in the blood. It is, perhaps, only in the two latter situations that we can submit it to minute anatomical investigation, and ascertain its peculiar structure and organization. When we examine anatomically a mass of carcinomatous matter contained in a large vein, or situated on the surface of a serous membrane, in loose cellular tissue, on the surface of a sore or cicatrix after the removal of an organ affected with Carcinoma, we find it composed of the following elements in various proportions, viz. carcinomatous matter; cellular, fibrous, and serous tissues; and bloodvessels.

The *carcinomatous matter*, whatever may be its consistence, almost always forms by far the greater bulk of the disease. If, however, its consistence be considerable, it generally presents a uniform, granular, or radiated, and, when soft, a lobulated arrangement. These three varieties are sometimes met with in the same tumour, and indicate the progressive development of the disease; the radiated arrangement being seen at the basis, the uniform and the lobulated towards the circumference of the tumour.

The *cellular tissue* is often small in quantity, and sometimes so fine and loose as not to be perceptible till after the carcinomatous matter has been separated from it by pressure and maceration. It encloses that matter, separates it into granules, bundles, or lobules; intersects these in various directions, and serves to conduct the vessels which administer to the nutrition and growth of the disease.

The *fibrous tissue* is not often met with as an anatomical element of Carcinoma in the situations in which we are now considering this disease. The *serous tissue*, on the contrary, is frequently present, and may form either a capsule to the carcinomatous matter, which is then said to be encysted, or give rise to the formation of cysts of various sizes containing gelatinous, albuminous, or other fluids.

When the carcinomatous matter is deposited in the molecular structure, instead of on the surface of organs, where we have just been considering it, the quantity of the cellular and fibrous tissues which intersect it in various directions is sometimes very

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considerable. In Carcinoma of dense organs, such as the breast, uterus, ovaries, liver, walls of the stomach, &c. these tissues are also often very abundant. Indeed, in the early stage of Carcinoma of these organs, a firm, pale, compact, cellulo-fibrous looking tissue is, not unfrequently, the only anatomical element discoverable, and which, on this account, and from the increase of bulk with which it is accompanied, has been described by Andral as *hypertrophy* of the cellular tissue,—an appellation which does not appear to me to be warranted either by analogy or by the changes which this tissue subsequently undergoes. For hypertrophied cellular tissue, such as we find in *Elephantiasis Arabum* or *Barbadoes Leg*, has no tendency to terminate in Carcinoma; nor does hypertrophy of the heart from disease, or of the muscles of voluntary motion from frequent exercise, ever present any other change than that implied by this term, except a certain increase of density, generally in proportion to the increase of bulk which has taken place. Besides, admitting that a certain degree of hypertrophy may precede the presence of Carcinoma, the facts which I have already brought forward in illustration of the mode of formation of the disease,—such as the deposition of the carcinomatous matter in the molecular structure of organs, its effusion on the free surface of serous membranes, and its separation from the blood contained in its proper vessels,—clearly show that no such change is necessary, inasmuch as the disease frequently occurs in situations in which this tissue is either small in quantity or does not exist. What, therefore, appears to be hypertrophied cellular tissue must be regarded as a tissue *sui generis*, produced by the uniform distribution and molecular deposition of the carcinomatous matter, either in the cellular tissue of an organ, or in an accidental tissue of a similar kind, formed during the deposition of the carcinomatous matter. Such is, in fact, the manner in which the cellular and fibrous tissues which enter into the composition of this matter are generally formed. These tissues are most conspicuous in the early stage of the disease, becoming gradually less apparent as it advances, and ultimately disappearing in consequence of their undergoing the carcinomatous transformation, or other changes afterwards to be noticed.

The *bloodvessels* which enter into the composition of the carcinomatous matter vary greatly in number, and sometimes considerably in bulk. They are rarely perceptible in any of the varieties of Scirrhus; are generally few in number in the first and second varieties of Cephaloma (the Organized and Mammary Sarcoma of Abernethy); but in the Medullary Sarcoma they are often so numerous as to form the greater portion of the brain-like tumour in which they ramify. When these vessels are examined in Cephaloma, they are found to vary in diameter from the breadth of a hair to a line, and present that peculiarity of distribution always more or less conspicuous in newly formed bloodvessels, that is to say, the ramifications of which they are composed communicate with a common trunk at its opposite extremities in the same manner as the hepatic and abdominal divisions of the vena portæ do with the trunk of this vessel. They are frequently varicose; their walls are remarkably delicate; and they have altogether much more a venous than arterial character. They appear to be formed apart from the vascular system of the surrounding tissues, as they can be seen to originate in small red points situated at the centre or at the circumference of the carcinomatous mass, which, at first, assume the appearance of striæ or slender streaks of blood, and afterwards acquire a cylindrical arrangement and ramiform distribution, thereby constituting what may be denominated the *proper circulation* of Cephaloma. The communication which exists between these vessels and those of the organ in which the cephalomatous substance is contained, is frequently very imperfect,—a circumstance which, together with the delicacy of their structure,

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renders them extremely liable to congestion and rupture. The most minute divisions of these vessels terminate by penicillated extremities in the carcinomatous matter, where they communicate with veins and arteries belonging to the affected organ. The latter vessels, which may be said to form the *collateral circulation* of Cephaloma, are seldom so conspicuous as the former, but there are cases in which they appear to constitute the greater part of the vascular structure of the disease. They proceed in a radiating direction from the pedunculated attachment of a tumour, for example, or arise along its circumference in the cellular tissue which separates it from the neighbouring parts. It is by means of these vessels that the materials required for the nutrition and growth of such tumours are supplied; and, as we shall see afterwards, the partial or even the complete destruction of these and other tumours similarly situated, is occasioned by causes which interrupt this their collateral circulation.

The bloodvessels which are seen in Schirroma appear to be no other than branches of those which belong to the neighbouring tissues, and which have become enclosed within the substance of which the several varieties of this species of Carcinoma are composed.

Physiological Characters of Carcinoma.—The anatomical characters just described are the most unequivocal circumstances by means of which we are enabled to perceive the existence and estimate the degree of those properties termed vital or physiological, which manifest themselves during the development of Carcinoma. But it is more especially the formation of cellular tissue and bloodvessels in the carcinomatous matter, which shows it to be in possession of these properties. We have already seen that the functions of circulation and nutrition are actively carried on in the carcinomatous matter. Of these two functions, that of circulation is by far the most important, inasmuch as many of the more remarkable phenomena which present themselves during the progress of carcinomatous formations, depend on changes which take place either in the proper or collateral circulation which I have described. Thus the quantity of blood contained in a carcinomatous tumour, and consequently various shades of colour of the substance of which it is composed, will depend much on the degree of facility with which the circulation is performed in either or both systems of vessels. An imperfect communication between these vessels, owing to the manner in which they are connected, or the presence of a mechanical obstacle in the situation of the collateral veins preventing the return of the venous blood, frequently give rise to congestion of the whole or a portion of a carcinomatous tumour, the colour of which becomes more or less red, purple, brown, or black. The congestion thus produced may be such as to give rise to rupture of the vessels, and internal or external hemorrhage. In the former case the carcinomatous substance, when situated externally in the form of a tumour, is seen to acquire a rapid increase of bulk proportioned to the extent of the effusion, and, when examined afterwards, is found to be infiltrated with blood, or broken down and mixed with clots of this fluid, and irregular masses or layers of fibrine, thereby producing, when the tumour possesses the cerebriform character, appearances very similar to those observed in cerebral apoplexy from sanguineous effusion. If the obstacle interrupt entirely the circulation in the tumour, nutrition ceases, and death ensues in all those parts of it from which the obstructed vessels proceeded. The termination of Carcinoma in mortification from obliteration of veins is far from being a rare occurrence. It sometimes occurs in whole tumours, but is most frequently observed in portions of them, or in some of the small tumours of which larger ones are frequently composed, that are attached by narrow pedunculated extremities. The unequal development of one of these small

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tumours may give rise to compression of a neighbouring one; or the tissue to which they are attached may, from its unyielding nature, act as a ligature on their pedunculated extremities, and intercept the return of the venous blood through them. The same thing sometimes happens to tumours that have perforated fasciæ. The protruding portion, now relieved from the pressure to which it was before subjected, increases rapidly in bulk; but the dimensions of the opening through which it passed remaining the same, a degree of constriction is produced which arrests the circulation through its vessels, when it dies and sloughs. Hence the delusive hope that nature had effected a cure of the disease, not only on account of the diminution of bulk, but also the imperfect cicatrization which sometimes follows the sloughing process. It is on the principle of diminishing the supply of blood for the nutrition and growth of these tumours, that the frequent local abstraction of this fluid, the application of cold, the use of the ligature and compression, have been recommended as the most effectual means of retarding or arresting their progress.

Changes similar to those I have just described, result likewise from the presence of the carcinomatous substance acting as a stimulus, and exciting various degrees of congestion. In consequence of the congestion thus produced, and the modification of nutrition which necessarily follows, softening also takes place not only of the carcinomatous substance, but likewise of the tissues which enter into its composition. Softening of this kind is sometimes effected with great rapidity, and tumours which before felt firm or even hard, acquire a soft pulpy feel, and, when laid open, are found to contain a fluid of the consistence of cream, intermixed with shreds of cellular tissue, detached bloodvessels, blood, and sometimes pus. This process of softening and sloughing is frequently seen taking place in carcinomatous tumours that have perforated the skin; and, when considered in connexion with the state of the circulation which has given rise to it, enables us to explain the peculiar appearance of those frightful solutions of continuity by which it is followed, such as their projecting everted edges, and rugged central excavation. It is well known that it is the most projecting part of a tumour situated beneath the skin, in which a solution of continuity commences; and the reason of this is, that it is here the circulation is first arrested from the greater degree of compression to which the bloodvessels are subjected, together with the increased influx of blood caused by a greater degree of irritation. The most elevated portion of the skin becomes atrophied during the first stage of compression and irritation, that is, when the circulation of the blood through it is only impeded; but so soon as this all-important function has ceased, which is announced by a change of colour from bright to dark red, purple, or black, a diminution of sensibility and temperature, it begins to soften, soon sloughs and exposes the subjacent portion of the tumour, whose circulation had been similarly modified, softened, and deprived of vitality to a greater or less depth. The edges of the solution of continuity of the skin when first formed are sharp and irregular; they are not *everted*; they are, on the contrary, sometimes *inverted*; and their thickness is in proportion to the depth of the slough. The peculiarity of form assigned to the edges is produced by the subsequent development of the carcinomatous substance situated beneath them, which, being entirely freed from pressure all round their internal margin, necessarily projects forwards, as it grows, towards the centre of the tumour hollowed out by the softening and sloughing process, and, consequently, carries them gradually upwards and backwards. They acquire, at the same time, a great accession of bulk, and form a rounded undulating border, beneath which the skin is found doubled upon itself, encircling the carcinomatous excavation.

[*Carcinoma*, 4.]

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All these changes which I have described, viz. congestion, hemorrhage, softening, and sloughing, take place in both species of Carcinoma. In Scirrhomia, however, they originate in the vascular system of the tissues included within the carcinomatous matter, but are not on that account less frequent and destructive than those which arise in the proper and collateral circulation in Cephaloma. In general the softening is less complete, the hemorrhage not so considerable, and the sloughing more extensive in the former than in the latter.

Softening may take place in any part of a carcinomatous tumour, although it has been maintained that the central portion is the primary seat of this change. Instead of being softer, the centre of the tumour is often much harder than any other portion of it. In such cases it consists of a nucleus of firm, grey, semi-transparent substance and obliterated bloodvessels, forming a central depression, around which the rest of the tumour presents a radiating structure. The depression is not observed unless when the tumour is divided or is situated on the surface of an organ, as the liver, where tumours of this kind are generally met with. In the former case the depression arises from the softer substance, after the division of the tumour, raising itself by its elasticity above the unyielding nucleus; in the latter it is produced by the peritoneum adhering to the surface of the tumour when small, and preventing its development in that direction. If the tumour does not come in contact with the peritoneum until it has acquired a considerable size, it presents no such depression, or only a very small one. Hence the reason why, in carcinoma of the liver, we meet with some tumours having a smooth globular surface, and others with a central depression of greater or less extent.

Nerves have never been detected in any of the varieties of Carcinoma as a *new formation*. They are sometimes included within agglomerated tumours, or even in a single tumour that has happened to form in a situation through which they pass. It is on this account that some pathologists have supposed the carcinomatous substance to be provided with nerves; and M. Maunoir, of Geneva, hazarded the opinion that Cephaloma, no doubt from its frequently resembling the substance of the brain, is in reality this substance effused by the nerves when under the influence of some peculiar morbid state; an opinion, to the accuracy of which the facts related in the preceding pages do not leave even the semblance of probability.

The last circumstance connected with the pathological anatomy of Carcinoma to which I shall allude, is the development of the subcutaneous venous system, sometimes so conspicuous when the disease affects the breast in the form of a tumour, or any other external part where the skin is capable of considerable extension. The dilated and varicose state of the subcutaneous veins in these cases is simply the consequence of the mechanical obstacle occasioned by the tumour to the venous circulation in its vicinity, and not the result of any special influence exercised by the disease. It is produced by tumours of every description similarly situated—fatty tumours and even cysts, and cannot, therefore, be considered as furnishing any evidence of the existence of Carcinoma in particular.

DESCRIPTION OF THE PLATES.

PLATE I.

In this plate are exemplified the several varieties of Scirrhone, viz. Scirrhous, Pancreatic Sarcoma, Lardaceous Tissue, and the Gelatiniform Cancer. *Fig. 1*, scirrhous of the breast. A, section of the scirrhous tumours, in which the bluish-grey, semi-transparent substance is seen separated into irregular masses, and intersected in various directions by the dull white or straw-coloured fibrous tissue. It is surrounded by fat, B, between the lobules of which the scirrhous substance projects in the direction of the cellular tissue. The skin, as is generally the case in tumours of this size, is in close contact with the scirrhous substance; and the nipple, E, although small, preserves its natural situation. The *depression* of this body arises in consequence of its being held down by the laciferous ducts which terminate in it, on the one hand, and on the other, of the increasing development of the scirrhomatous deposit around it. But the depression is never very marked until the tumour has become fixed in the direction of the ribs, and therefore its presence must always be regarded as an unfavourable sign. *Fig. 2*, 3, and 4 are specimens of the Pancreatic Sarcoma. *Fig. 1* represents a section of two tumours, AE, of this kind situated in the breast, the first of which equals in bulk an orange, and is composed of a multitude of lobules, formed of a congeries of smaller ones, separated by laminated cellular tissue, and enclosed in a general capsule, D, of the same tissue, having in some parts, B, the characters of a serous membrane and a cystiform arrangement. In *fig. 3* we have a view of the same tumour carefully dissected, in order to shew that the pancreatic sarcoma is frequently made up of agglomerated, pyriform tumours, AAA, a transverse section of which gives rise to the lobulated appearance of the cut surface of the general tumour. Between the pyriform tumours and the fat, D, is situated a small portion of the general tumour, B, towards which the former converge, and terminate in the cellular tissue and remaining healthy substance of the gland. The collateral circulation, CC, of several of the pyriform tumours is in a state of considerable congestion, and in several points hemorrhage has taken place. *Fig. 4* is a section of a pancreatic tumour situated between the breast and axilla, and presents the appearances most frequently met with in this kind of tumour. It has altogether the lobulated structure, colour, and consistence of the pancreas. A, the lobulated cut surface; B, the cellulo-fibrous capsule of the tumour. *Fig. 5*, 6, and 7 represent the lardaceous variety of scirrhoma in the lungs, uterus, and breast. Almost the whole of the left

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lung, of which *fig. 5* represents a portion, was converted into a dense substance resembling a section of fresh pork. The lobular structure, however, of the organ, AA, was very conspicuous; but the bloodvessels and bronchi were either greatly compressed or obliterated. Towards the upper extremity of the lung, BB, the carcinomatous deposit is seen extending from lobule to lobule, and at CC has made its way through the bronchi. The pleura costalis and pulmonalis were studded with tumours of the same kind, varying from the size of a pin's head to that of a walnut. Several of these tumours are seen at D, on the pleura pulmonalis, the largest divided, and arising by a broad basis; the others entire, round or pyriform, attached by a peduncle. *Fig. 6* is a remarkable example of this variety of scirrhus of the os tinctæ. A, neck of the uterus; B, vagina; C, the external surface of the lardaceous substance, which was entirely confined to the os tinctæ. A section of this substance is seen at D, which is about half an inch in thickness, and terminates abruptly in the healthy tissue of the neck. The patient was about forty years of age, and had the disease removed nearly five years ago by Mons. Roux, and, so far as I am aware, has had no return of it since. *Fig. 7* represents a tumour, A, of the same kind in the breast, but undergoing the process of softening, as seen at BB, where a creamy-looking fluid has been forced out by pressure on the tumour. The presence of this fluid, which may resemble milk or cream, in tumours, however doubtful may be their characters in other respects, may be regarded as the most decided evidence of their being of a carcinomatous nature. The congested state of the collateral circulation which precedes the softening process is marked at CC, with a tendency to hemorrhage in several other points. The tumour is seen surrounded with fat, EE, and connected at D and E with the pectoral muscle and cellular tissue. *Fig. 8* is a fine specimen of the gelatiniform cancer of the stomach and epiploon. A, duodenum; B, pylorus; C, D, E, cavity and walls of the pyloric portion of the stomach; F and G, the epiploon. In all these situations the *colloid* or *gelatiniform matter* is collected into rounded masses of various sizes, agglomerated together, and lodged in cells formed by a dense tissue, composed apparently of the degenerated tissues of the affected parts. In the substance of the walls, as well as on the surface, of the pyloric portion of the stomach, the adventitious deposit is recognized by its transparency and cystiform arrangement. Several of the cysts are seen divided, formed of two, three, or more lodges, and emptied of their contents. This disposition of the cysts is best seen in the epiploon at G, which, in this form of scirrhus, is always more or less contracted upwards and around the great curvature of the stomach, and having from one to two or three inches in thickness. The walls of the stomach likewise undergo a great increase of bulk, as is well exemplified in the present case. The peritoneal surface, D, is frequently studded with small round masses of the same deposit; and in some cases the lymphatics in the same situation are filled or more or less distended with it.

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PLATE II.

This plate contains examples of the varieties of Cephaloma, viz. the Common Vascular or Organized Sarcoma, Mammary Sarcoma, Medullary Sarcoma, and Fungus Hæmatodes. *Fig. 1* is, perhaps, a *unique* representation of the first variety. It formed a large ovoid tumour, situated on the internal and upper surface of the thigh. It was attached by a narrow neck, B, to the subcutaneous cellular tissue, the skin, A, covering it, being eroded and bloody, from the friction of the opposite thigh. When divided longitudinally, as represented in the plate at C, it had the appearance of firm coagulable lymph or pale fibrine, of a faint yellow or greyish tint and uniform aspect. In a few points only, D and EE, could a vascular organization be perceived, and in these it was both limited and imperfect, particularly at E, where the vessels were obviously of new formation. *Fig. 2* is another tumour, situated in the immediate vicinity of the former, but presenting the characters usually ascribed to scirrhus. A, the scirrhus structure, composed of the grey transparent substance and the surrounding opaque fibrous tissue; B, a small portion of it resembling the substance of the other tumour, and which may be considered as an example of the transition of the former into the latter, which I have said is frequently the case. C, the skin; and D, the cellular peduncle of the tumour. *Fig. 3* is a good example of the mammary sarcoma in the breast. The lobulated structure, AA, of the whole tumour is very conspicuous. The whole of the gland was more or less affected, but the lactiferous ducts, B, were still visible at its upper extremity. Bloodvessels are seen making their appearance in several of the lobules, and at C slight hemorrhage has taken place. The nipple, D, has preserved its natural situation, as is generally the case in this variety of cephaloma. The lymphatic glands, *fig. 4*, presented the same form of carcinoma, but in a more advanced state. Two of these glands, AA, were composed of the same substance as the tumour in the breast, with a slight degree of vascularity. A third, B, was composed of the mammary and cerebriform substances, with great vascularity and a tendency to hemorrhage; and a fourth, C, was entirely transformed into the latter substance, which contained a considerable quantity of effused blood,—thus furnishing a marked example of the transition of the mammary to medullary sarcoma and fungus hæmatodes in the same individual, and in those glands which are generally believed to become affected with the disease in consequence of absorption. *Figs. 5* and *6* are examples of medullary sarcoma in the cerebellum. In *fig. 5* the disease occupies nearly one-half of the left lobe of the cerebellum, A, in the form of a lobulated tumour, extending laterally and backwards in the direction of C and B. So similar was the substance of the tumour to that of the cerebellum, that it was impossible to distinguish the one from the other otherwise than by the difference of form and arrangement which it presented in each. The uniform lamellated arrangement of the cerebellum became gradually lost in the lobules of the tumour, which were very vascular and separated from one another by delicate cellular tissue. In *fig. 6* the tumour projects from the cerebellum, A, into the fourth ventricle, C, and is of the size of a hen's egg. It is represented cut longitudinally, and in each half, BB, a multitude of slender bloodvessels are seen ramifying in every direction; and, from the presence of the *arbor vitæ*, D, at its basis, it is seen to be continuous with the substance of the cerebellum. *Fig. 7*

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is a tumour of the same kind which is not unfrequently met with projecting into the cavity of the cranium from the cells of the sphenoid bone. It consists of a number of smaller tumours, AA, of various sizes, covered by the dura mater, composed of a cerebriform substance, with which the cells of this bone are filled. The extreme vascularity of the cerebriform matter, B, is very conspicuous in the cells of the bone CC, which are represented laid open by a vertical section.

PLATE III.

This plate represents the seat and some of the more remarkable forms of carcinoma. *Fig. 1* represents a portion of the peritoneum, to which were attached a multitude of carcinomatous tumours, varying from the size of a pin's head to that of a cherry or small orange, either single or grouped together, A. They were of a round, ovoid, or pyriform shape, attached by a broad basis, D, or a peduncle, B, C, of considerable length. Some of them were very red, others quite pale, and were either entirely composed of cerebriform matter, or of this and a cellulo-vascular tissue, D, in various proportions. The peritoneum appeared to be quite healthy. *Fig. 2* is one of the best examples I have met with of the presence of the carcinomatous matter in the veins of the stomach. A, the pylorus; BB, a large carcinomatous ulcer of the stomach, the everted and rounded edges of which were composed chiefly of cerebriform matter. C, one of the coronary veins (the other was in a similar state) distended with the same kind of matter, as well as several of its branches, DD and EE. All these branches arose in the vicinity of the ulcer, which occupied the internal surface of the organ, and those marked EE are represented arising in the submucous tissue, the peritoneal and muscular coats having been dissected away. Some of these branches appeared to be in connexion, at their extremities, with the cerebriform matter which formed the walls of the ulcer, while others arose from portions of the stomach in which none of this matter was present. The branches of the vein, FF, towards the fundus of the stomach contained coagulated blood, which became gradually pale and fibrinous the nearer it was examined to the cerebriform matter, with which it was ultimately confounded. In some parts of the trunk of the vein this matter was sufficiently firm to preserve its cylindrical form when cut transversely, as at G: in other portions it was quite fluid and not vascular. Although in *fig. 3* the *ramiform* disposition of the carcinomatous matter is not so well seen as in the former, it represents, however, one of those cases of carcinoma of the kidney to which I have several times alluded, in which this matter is contained entirely within the veins. AA, cortical substance of the kidney; BB, tubuli uriniferi; C, pelvis and ureter; D, artery; E, emulgent vein; F, a cylindrical mass of carcinomatous matter projecting into the vein and adhering to its walls by means of cellular tissue. The same matter occupies a great number of the branches, GGG, situated in the substance of the kidney, and is continuous with that seen in the trunk, as indicated by the two probes, HH. In the latter it was of a lardaceous and cerebriform consistence, and in some parts very vascular. In the former it presented also the same characters, but the cerebriform more generally prevailed. *Fig. 4* is a remarkable example of the cerebriform matter contained in the vena portæ and two of its branches. A, the vena portæ divided before it enters the liver; B, C, D, four of its principal branches. The carcinomatous matter is seen projecting at E into the vena portæ, and from thence

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stretching in a cylindrical form, F and G, into the splenic and mesenteric veins. It completely filled the cavity of these veins, lay merely in juxta-position with their lining membrane, and presented a perfectly smooth surface of a pale yellowish-brown colour. A transverse section of it is represented at H, where it is seen to be composed of cerebriform matter, throughout which a considerable number of minute bloodvessels are distributed, and having no connexion whatever with the general circulation. This matter became less apparent when examined in the direction of the branches, and appeared to pass almost imperceptibly into the fibrine and coagulated blood, F and G, contained in the splenic and mesenteric veins, at the distance of from two to three inches from its termination in the vena portæ. *Fig. 5* represents a section of a carcinomatous tumour, of which there were a great number of various sizes growing from the peritoneum. Some of them were composed chiefly of fluid and coagulated blood and fibrine, and others of fibrine, cerebriform matter, with here and there collections of blood of greater or less quantity. AA, irregular masses of fibrine and cerebriform matter. BB, the sanguineous collections. CC represent bloodvessels, which were very numerous and conspicuous. All these tumours were contained in a serous capsule, continuous with the peritoneum. *Fig. 6* is a good example of the *stratiform* arrangement of the carcinomatous matter under the pleura pulmonalis. A and B, large and small isolated patches of this substance; CC, patches uniting; and D, a section of a single patch, to show its superficial character.

P L A T E I V .

This plate contains examples of carcinoma as it occurs in the bones. *Fig. 1* represents a section of one-half of a large carcinomatous tumour which surrounded the superior third of the femur. A, the lobulated surface of the tumour inclosed within an expansion of the periosteum and condensed cellular tissue; B, the head of the femur; and CC, a section of the upper third of this bone. The cut surface of the tumour, DD, presented a pale yellowish-grey colour, particularly where it was connected with the surface of the bone, and assumed a rose-red tint where it terminated in the circumference EE. In the same manner did its consistence vary from that of cartilage or bone in the former situation, to that of fresh pork, boiled udder, or brain in the latter. The brain-like substance was confined exclusively to the circumference of the tumour, and where it was greatest in quantity, as at F, possessed a considerable degree of vascularity. Besides these appearances, there was also observed a beautiful fibriform radiated structure of almost the whole of the tumour. The fibres proceeded in bundles from the basis to the surface of the tumour, each containing a number of fine bony spiculi, from half an inch to two inches in length, arising from the surface of the femur: around these spiculi the firmer substance of the tumour was accumulated. The medullary canal, G, contained a considerable quantity of the carcinomatous matter of a lardaceous consistence accumulated in its cellular structure, which, superiorly, is still conspicuous. Inferiorly, at H, this structure has disappeared, and the lardaceous substance forms a homogeneous mass of considerable bulk. *Fig. 9* represents a portion of the femur deprived of the soft parts by maceration. A, the trochanter major; BB, the bony spiculi arising from the shaft of the bone. The texture of this part of the bone was spongy but firm. *Fig. 3*, a section of a similar tumour surrounding the inferior fourth of the femur. A, one of the condyles of the femur; B, the interior of this bone. The relations of this tumour,

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C, with the bone were the same as in the preceding case. The radiated and fibriform arrangement of its substance, so marked in the other tumour, was hardly perceptible, and it contained no bony spiculi. It was composed of the mammary and cerebriform substances; the latter, occupying chiefly the circumference, contained a considerable number of newly formed bloodvessels, and presented here and there a few hemorrhagic spots. Similar substances occupied the canal of the bone F to the extent of about three inches. Here the walls of the bone were somewhat soft, and perforated in a great number of points. The muscles passing in front of the tumour D were expanded over its surface in the form of a thin membrane, beneath which the colour of the substance of the tumour was readily perceived. An interesting circumstance in the history of this case was the occurrence of hemorrhage, E, and the presence of a considerable quantity of cerebriform matter in the effused blood between the muscular fibres. *Fig. 4* represents carcinoma confined to the interior of the femur, and giving rise to what is called *spontaneous* fracture. A, the head of the bone; B, trochanter major; C, the walls of the shaft divided longitudinally; D, the fracture, which is ragged and bloody; EE, masses of the carcinomatous substance tinged yellow with pus, as well as the medulla, F, in the head and neck of the bone, and the fat on its external surface. The substance of the bone in the situation of the fracture cut like cartilage.

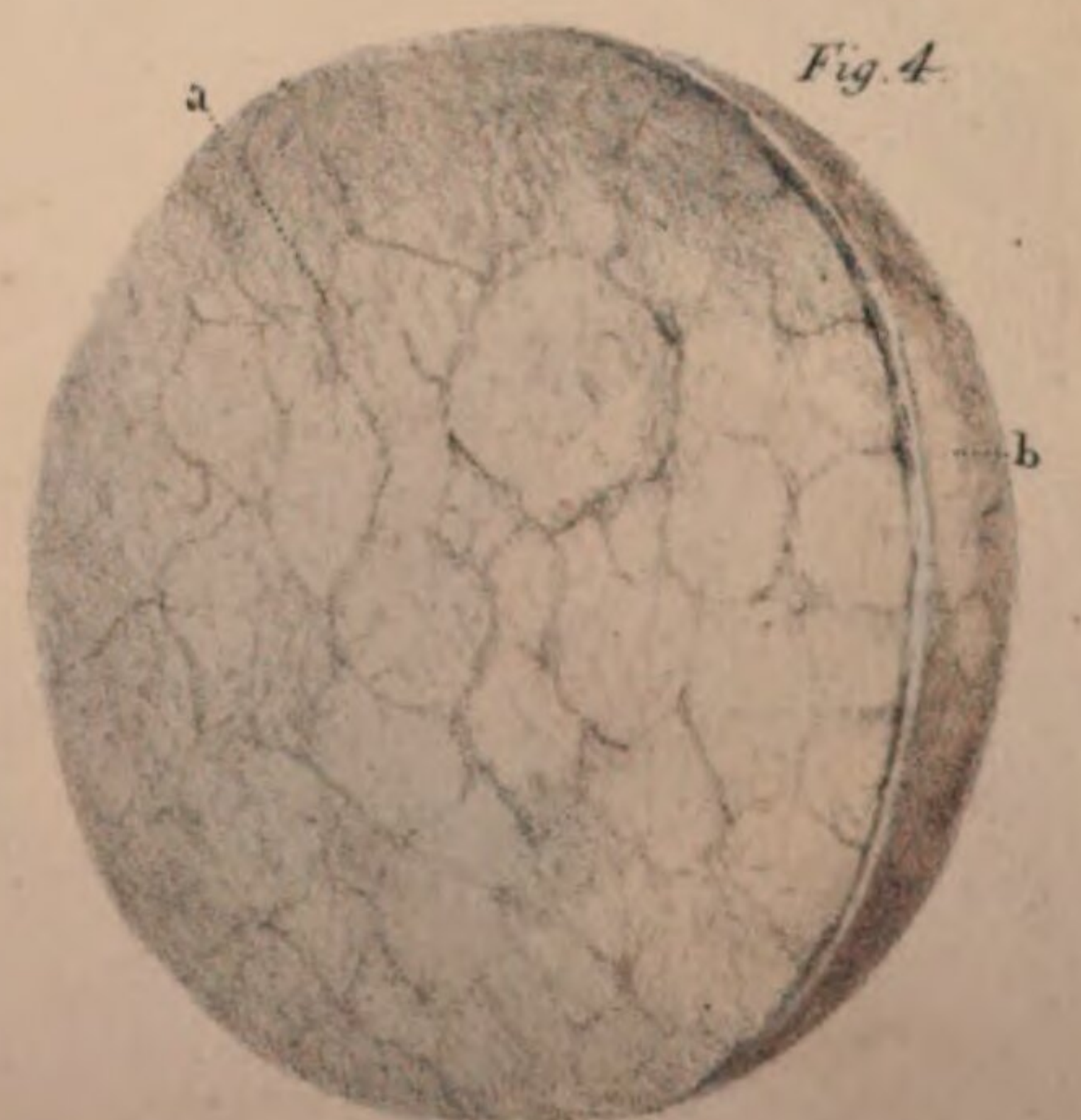
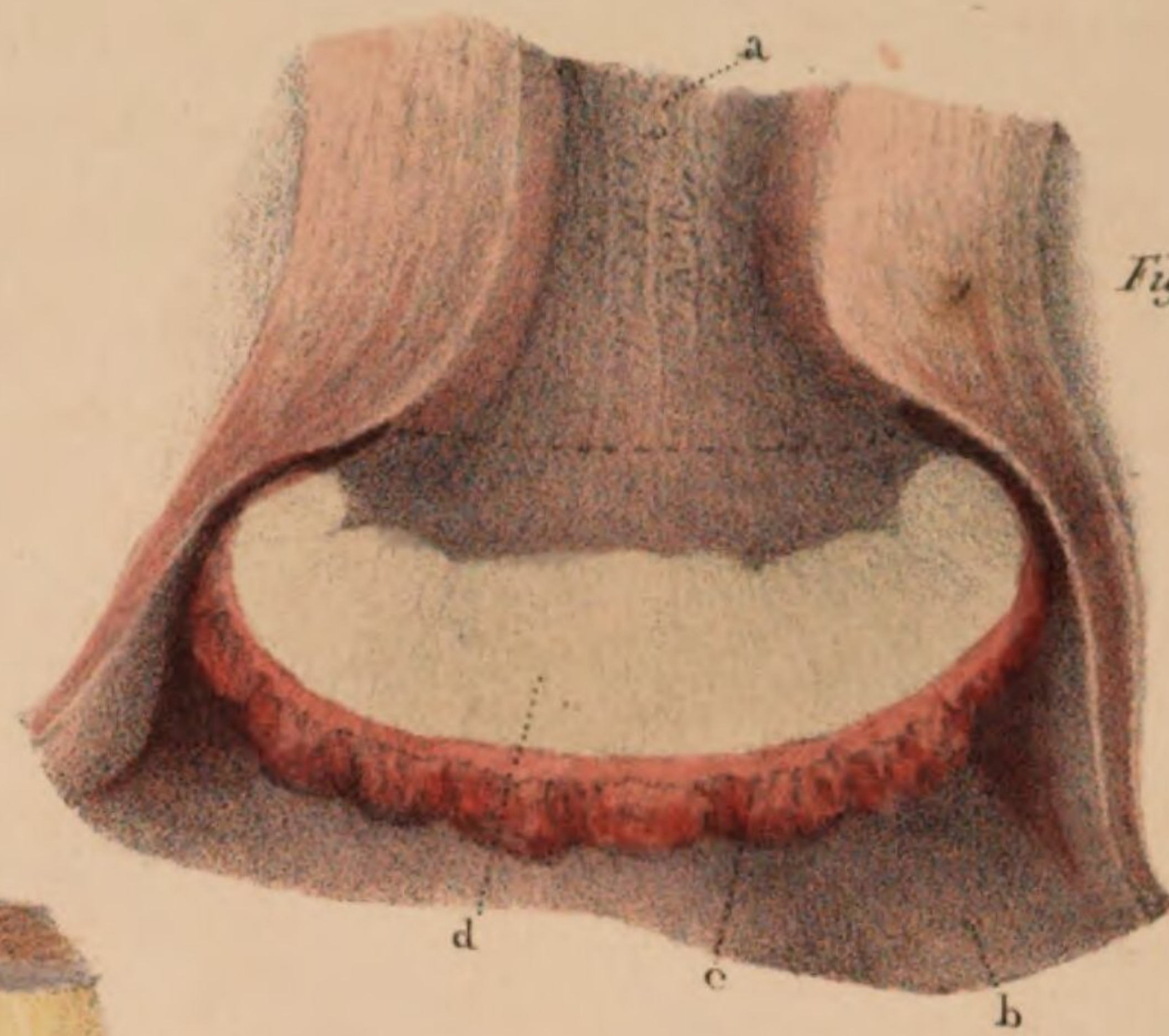
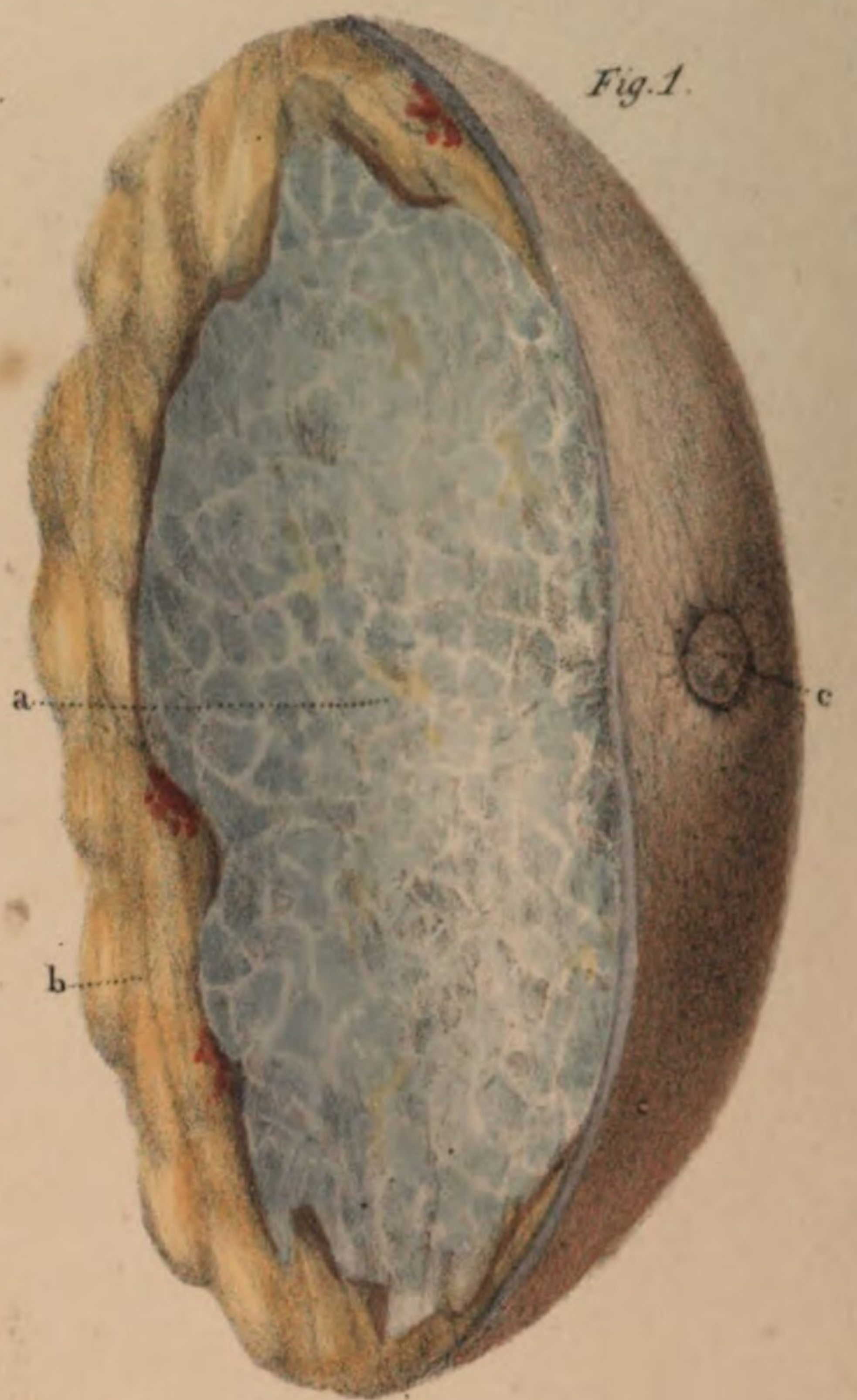
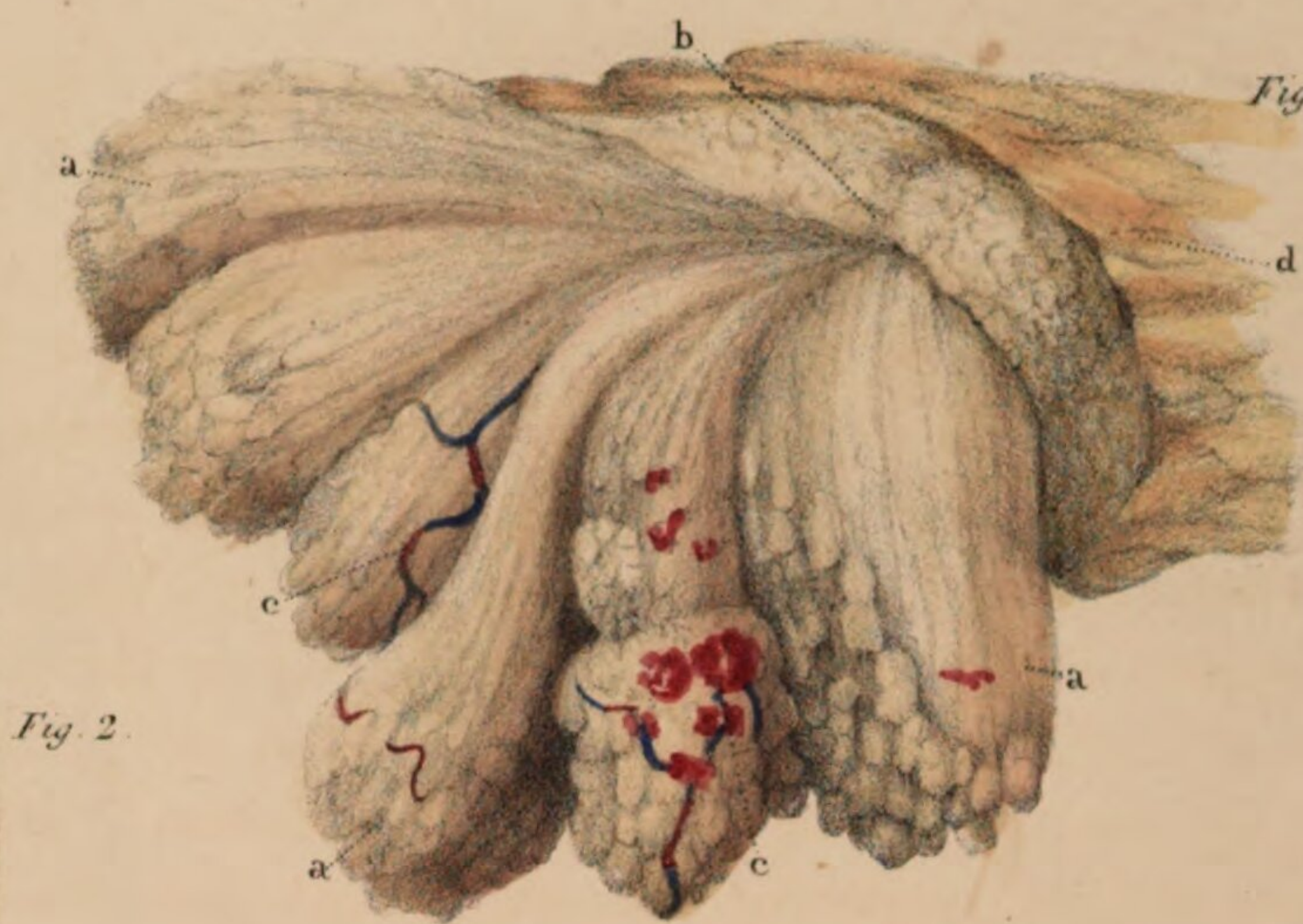
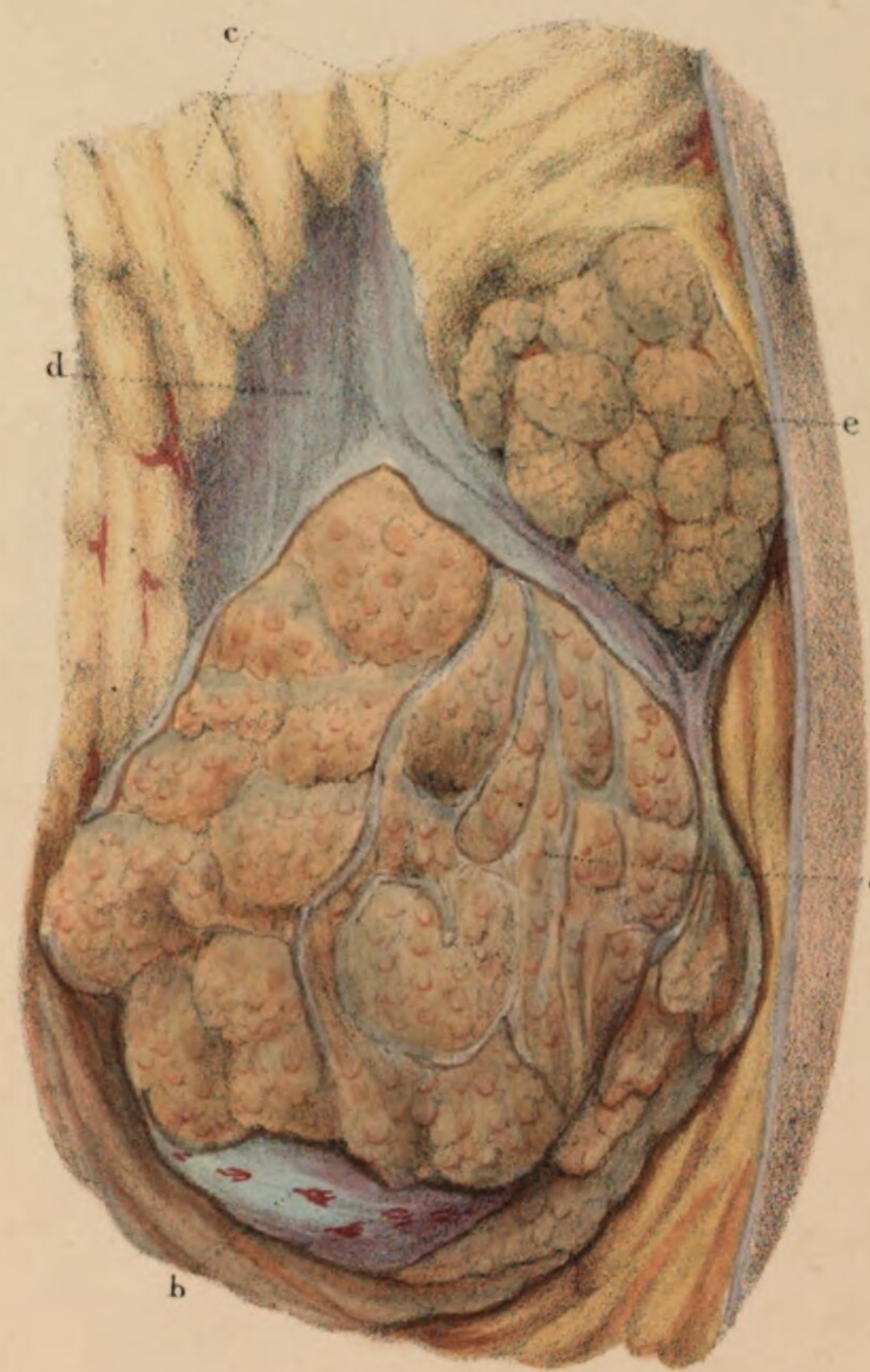


Plate II.

Fig. 1.

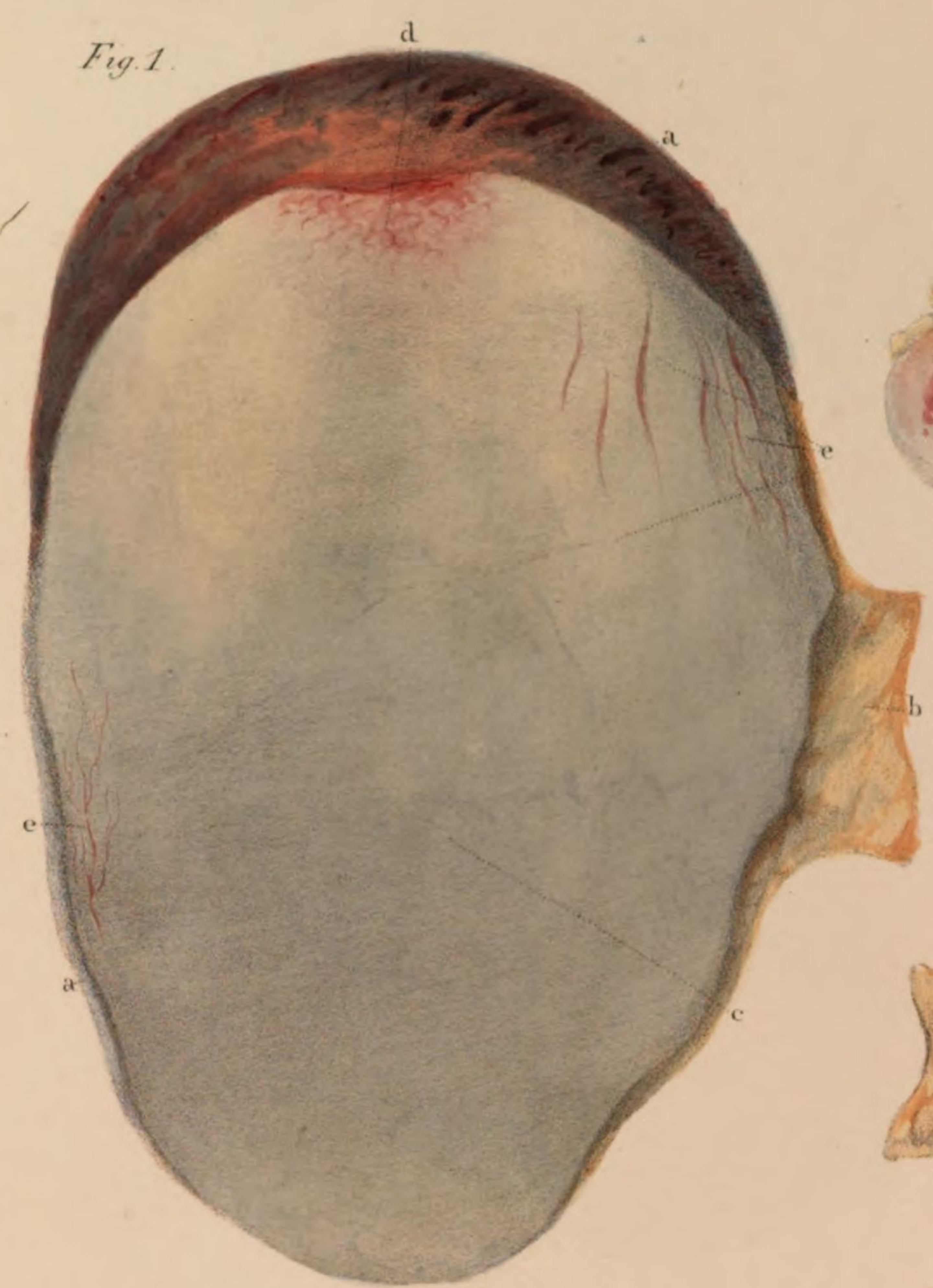


Fig. 4.



Fig. 3.

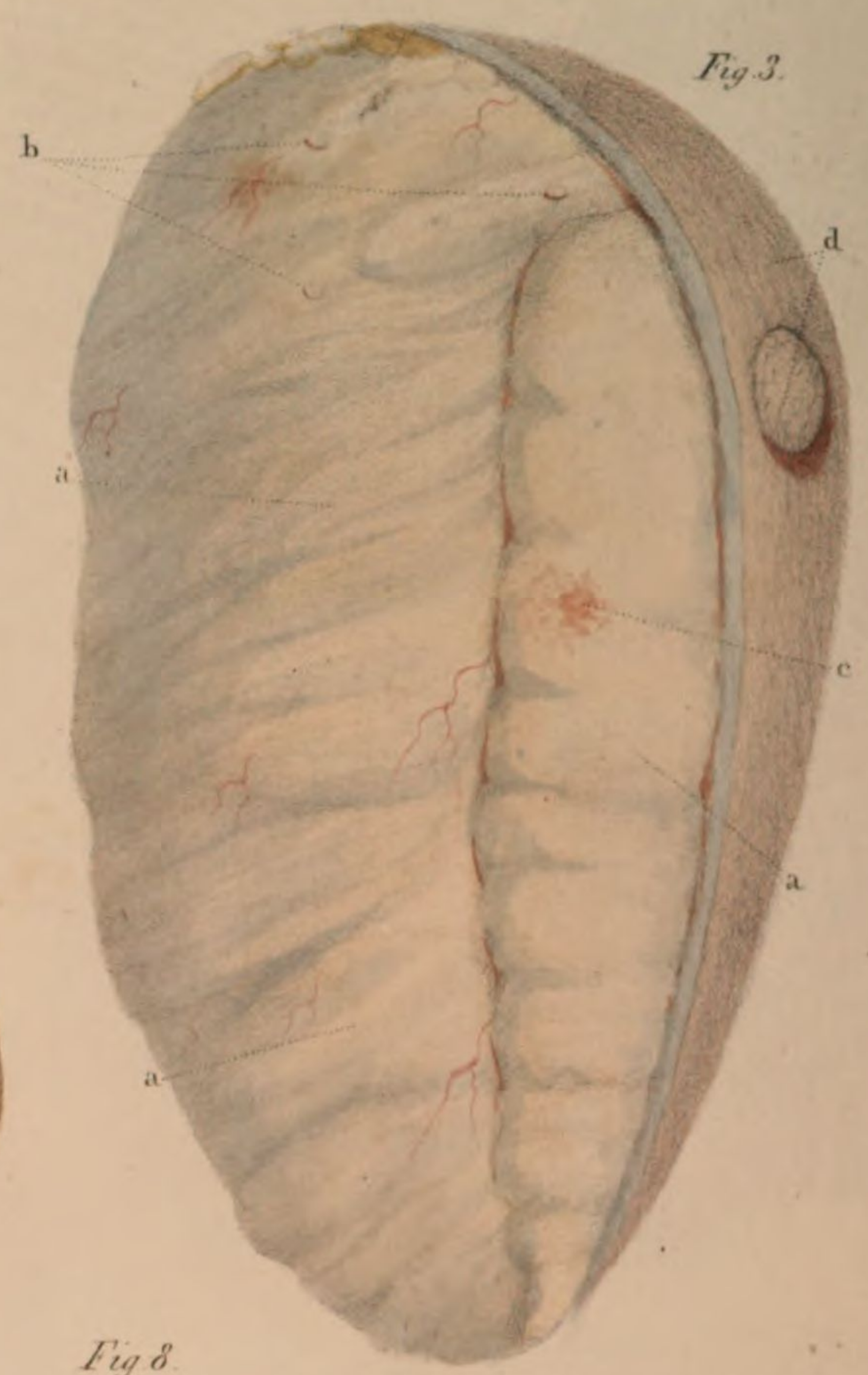


Fig. 2.



Fig. 8.



Fig. 6.

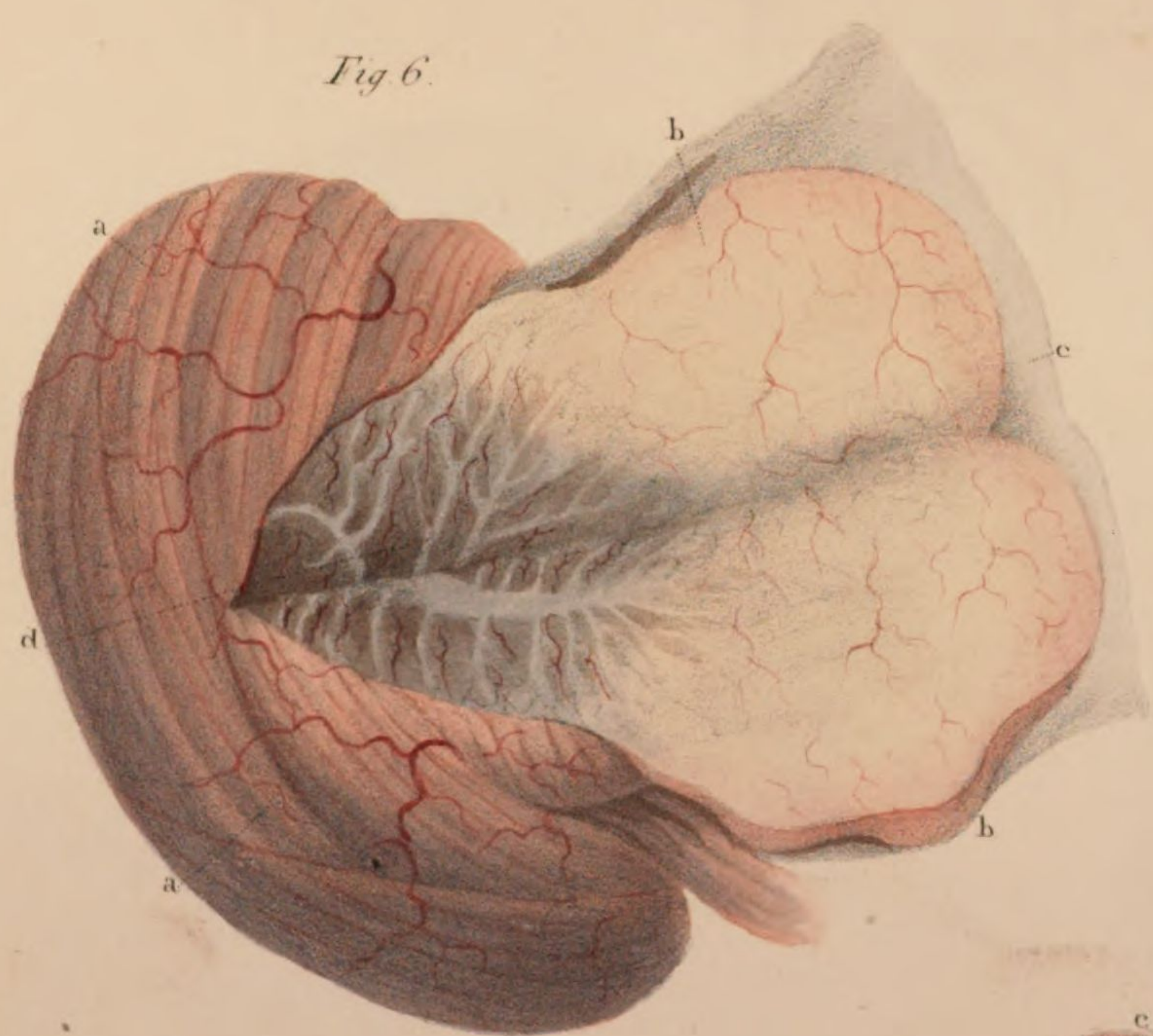


Fig. 5.

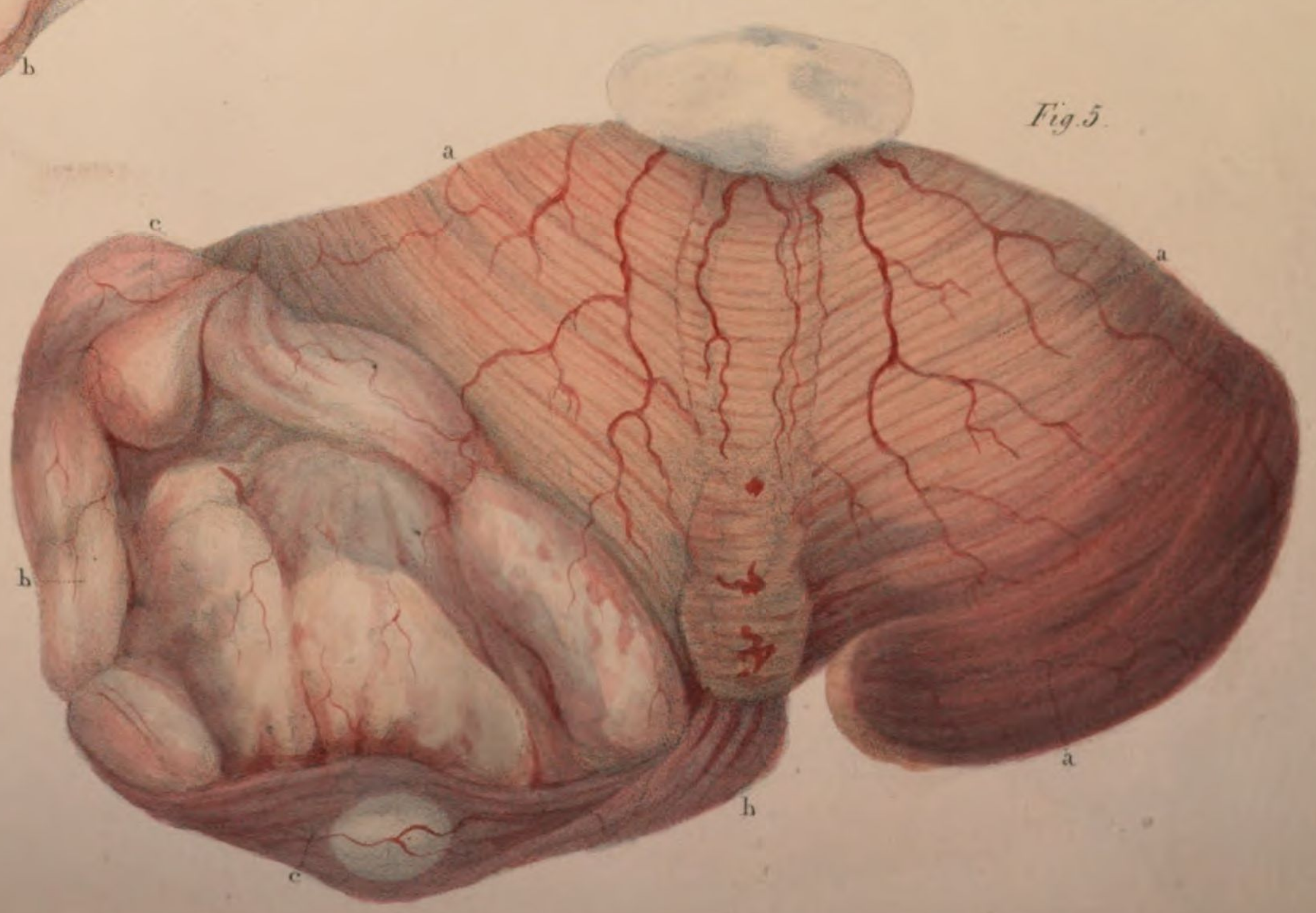


Fig. 7.

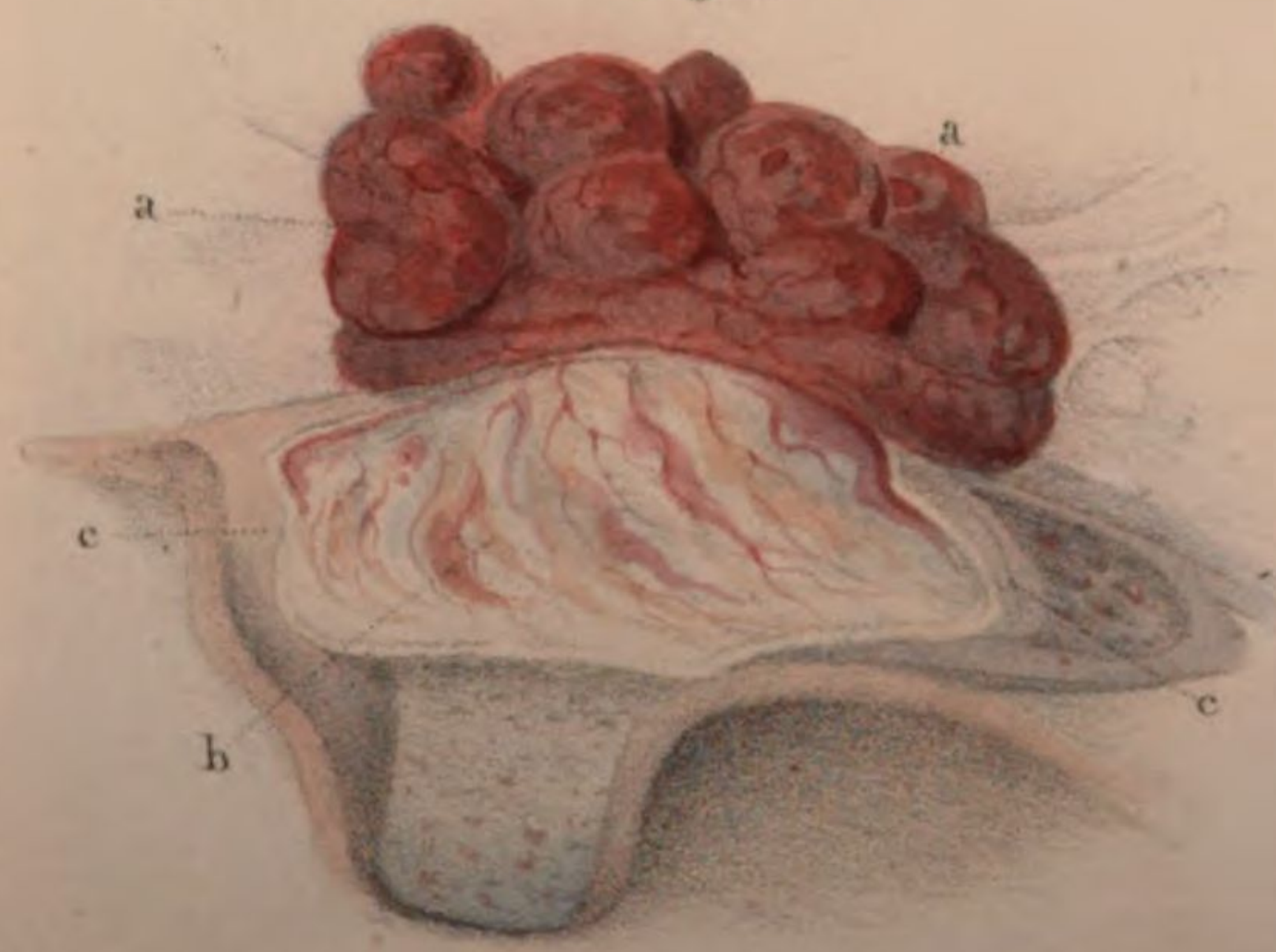


Plate III.

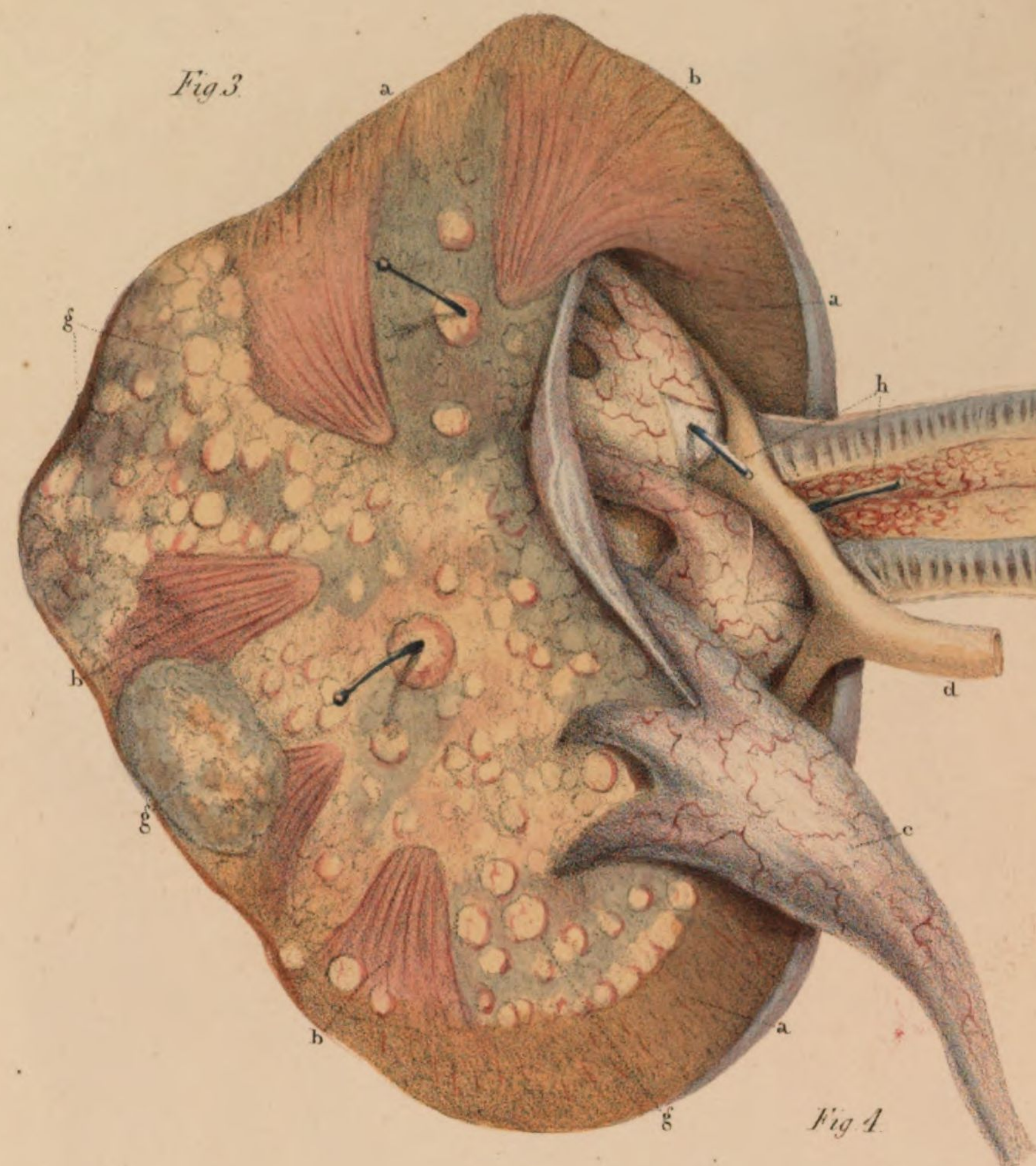


Fig. 1.



Fig. 4.



Fig. 5.



Fig. 6.

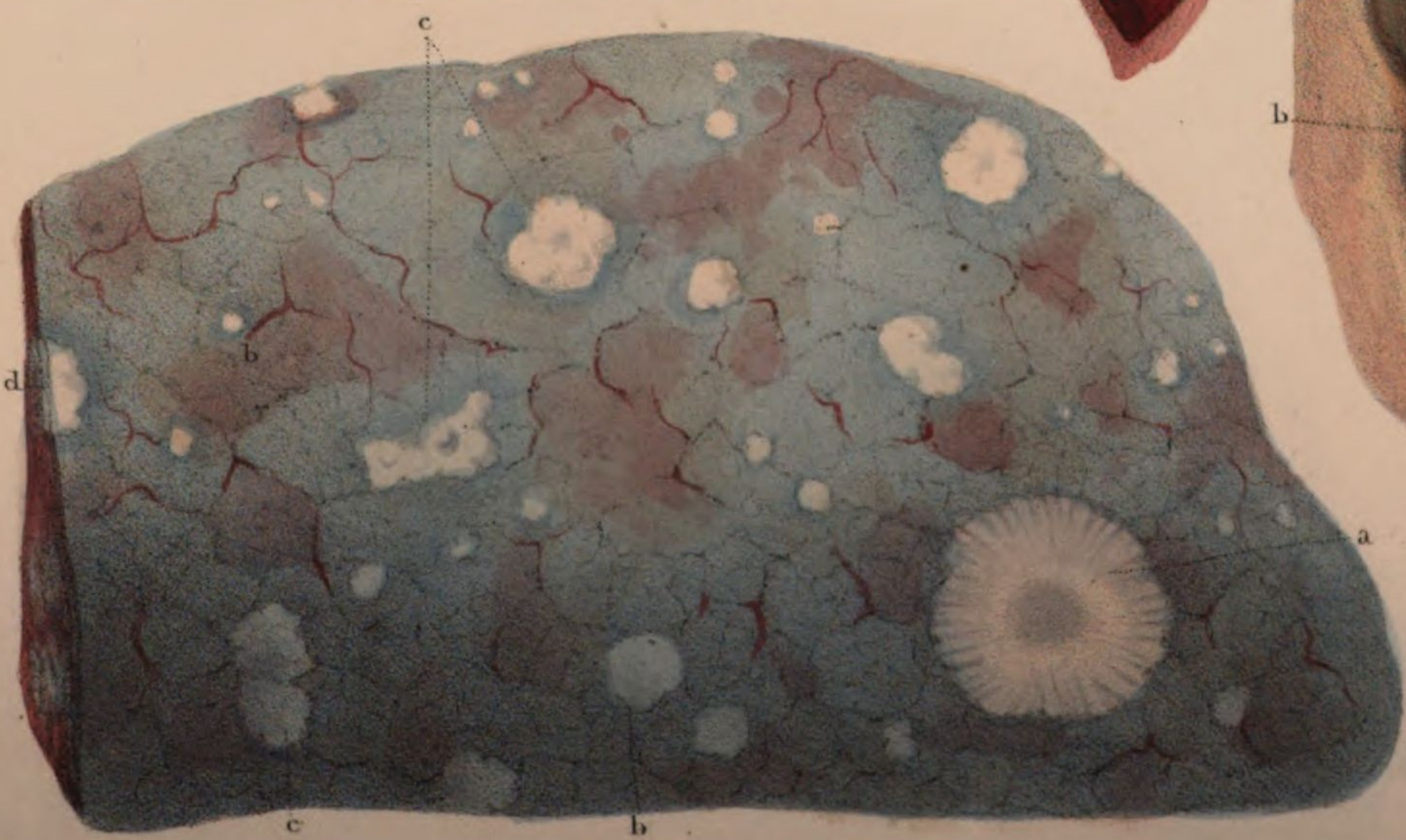


Fig. 2.

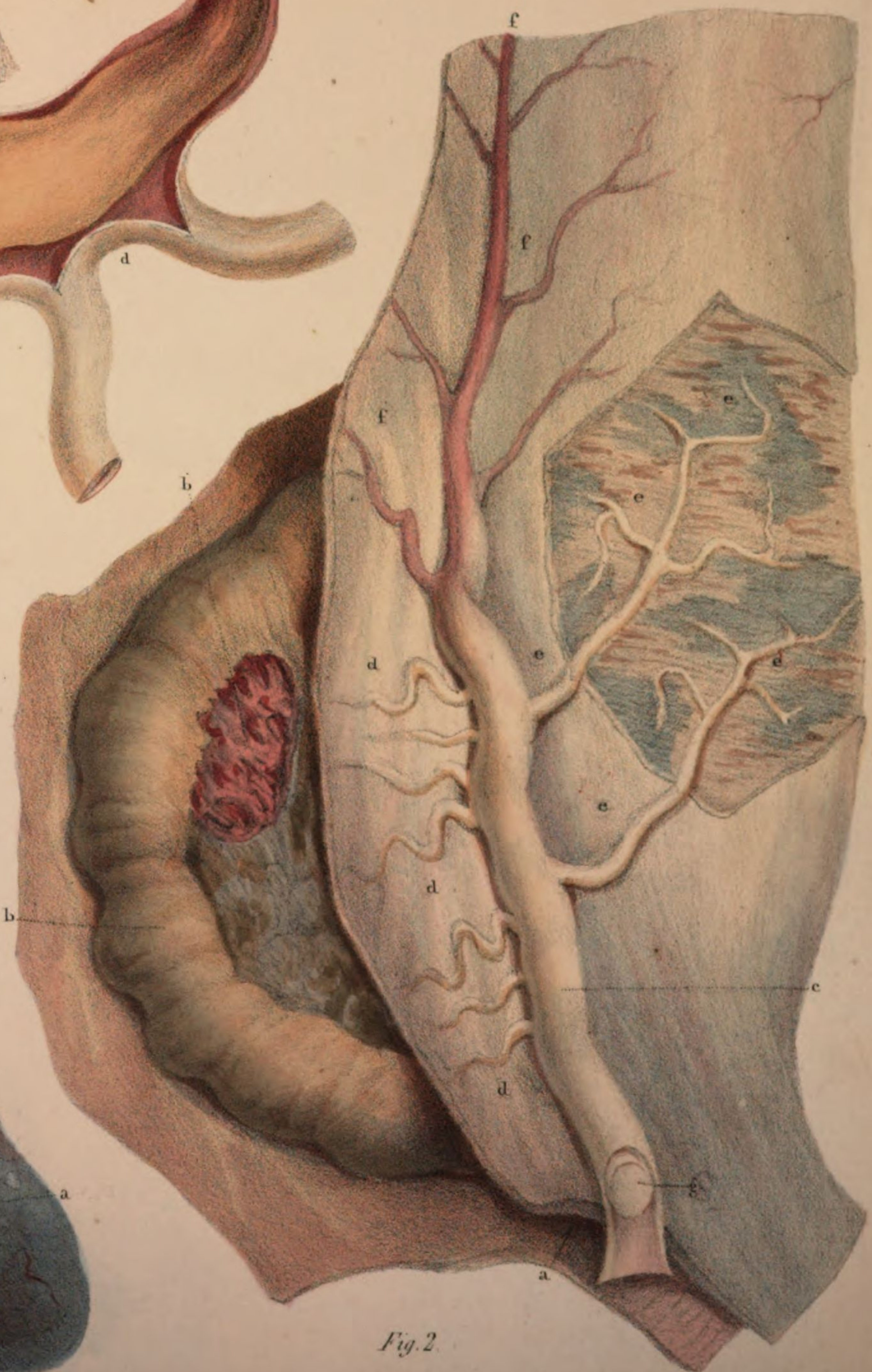
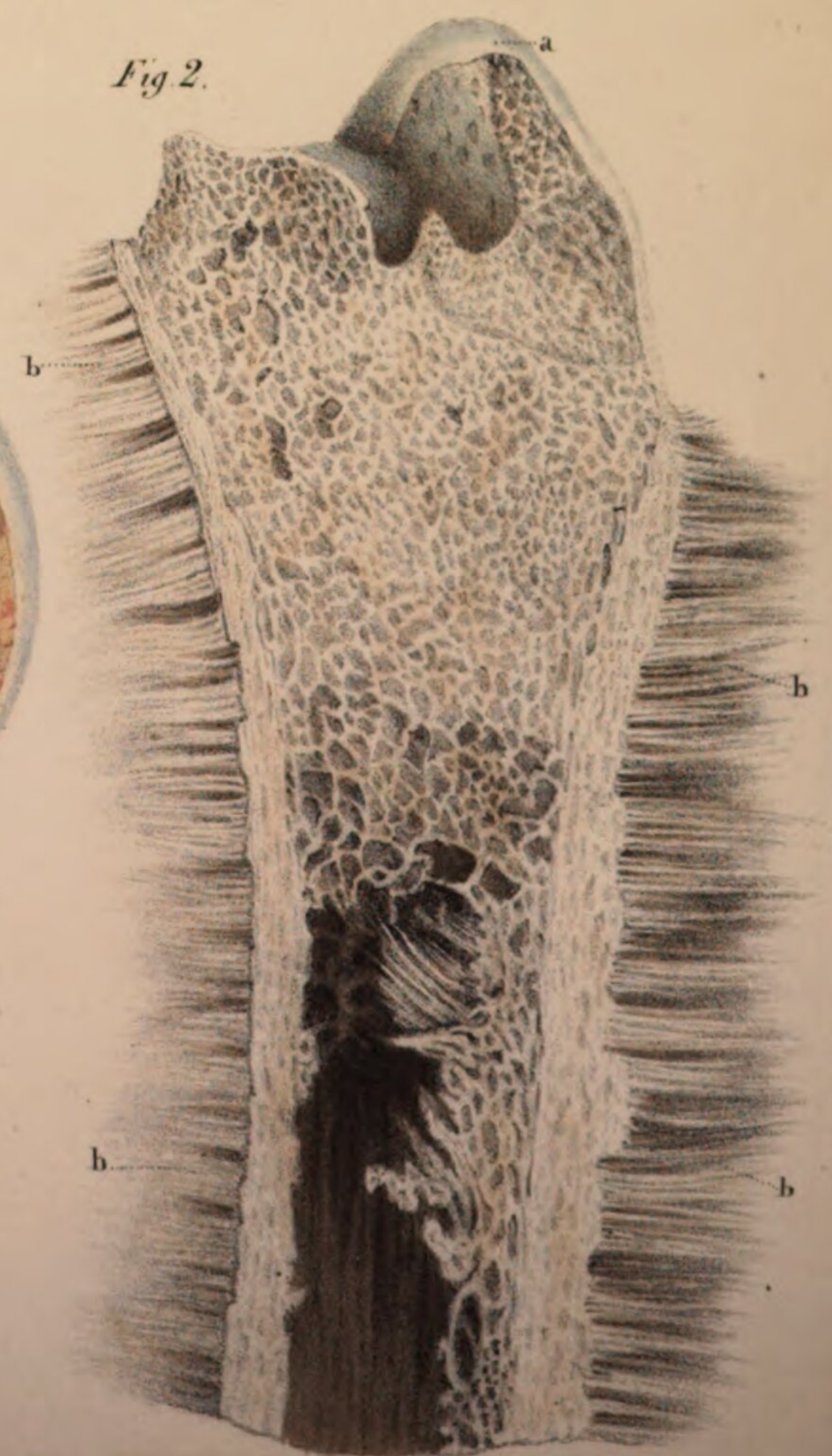
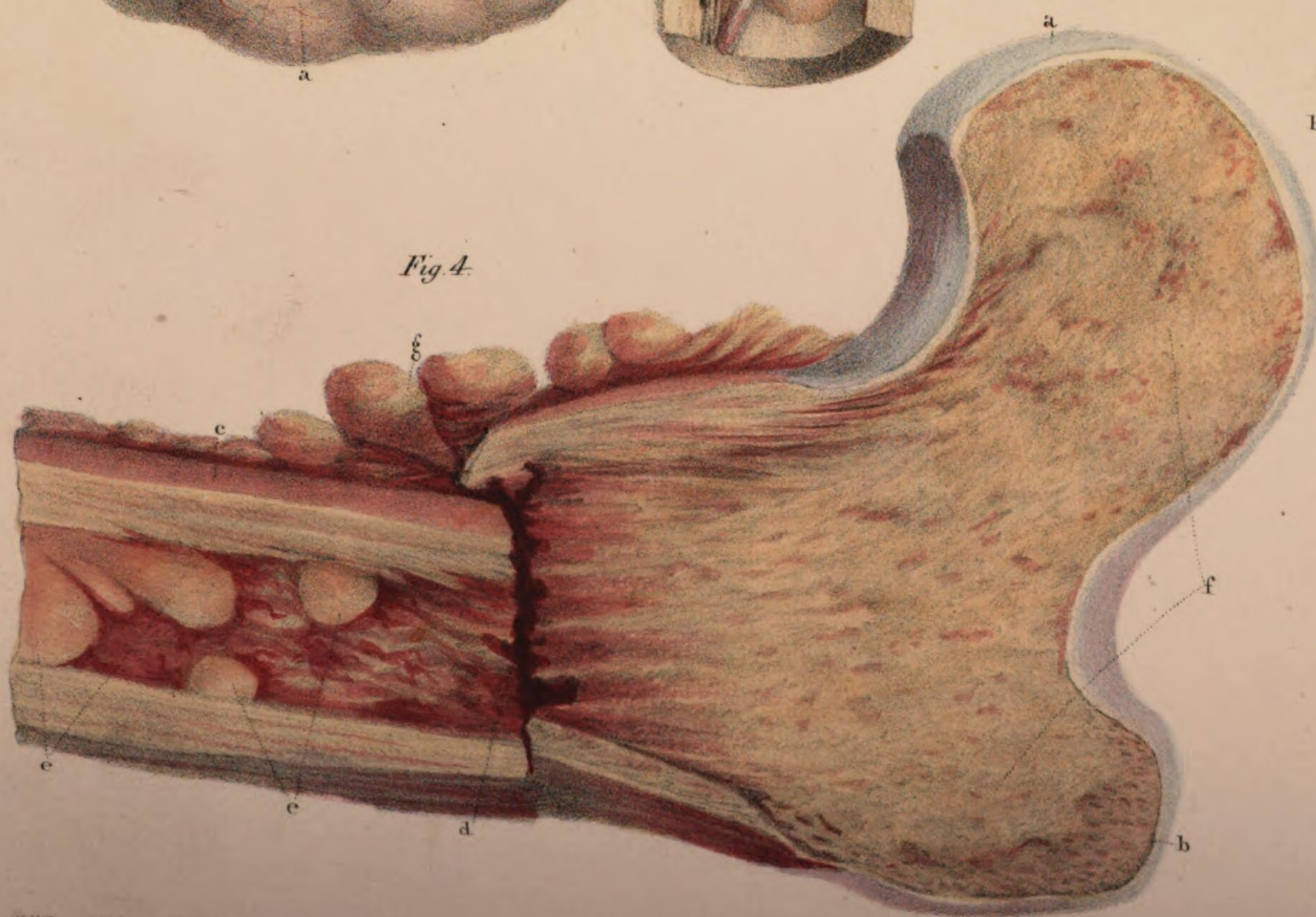


Plate IV.



TUBERCLE.

TUBERCLE.

THE term tubercle is employed to designate a peculiar morbid product, which pathologists describe as occurring in various organs in the form of a small round body, said by some to consist at first of a firm, grey, somewhat transparent, substance, which afterwards becomes opaque and of a dull yellow colour, and may then be broken down between the fingers like a morsel of cheese. These characters are said to represent what is called the first period, or crude state of tubercle. At some subsequent, but indefinite period, the crude tubercle loses its primitive consistence,—in virtue, it is believed, of certain inherent properties, by means of which it is converted into a liquid mass of the consistence of cream. It is further stated that this process, which constitutes the period of softening, is perceived always to take place in the centre of the crude tubercle, and proceeds from thence to its circumference.

Such is the description of tubercle given by Laennec, but which is considered by several eminent pathologists to be inaccurate.

Andral, in particular, describes tubercle at its origin as a pale yellow, opaque, small, round body, of various degrees of consistence, and in which no trace of organization or texture can be detected. He denies that the grey semi-transparent corpuscle or granulation described by Laennec, and since by Louis, constitutes the primitive state of tubercle, or that the process of softening takes place invariably in the centre of this substance. The latter change he ascribes to the admixture of pus secreted by the tissues subjected to the stimulus of tubercle as a foreign body, and not to any change originating in the tubercle itself.

Definition of Tubercle.—The following is, I conceive, a correct definition of tubercle, or rather of the tuberculous matter which constitutes the essential anatomical character of those diseases to which the term tubercular is now exclusively restricted.

Tuberculous matter is a pale yellow, or yellowish-grey, opaque, unorganized substance, the form, consistence, and composition of which, vary with the nature of the part in which it is formed, and the period at which it is examined.

To comprehend fully the manner in which the latter circumstances modify the physical characters of tuberculous matter, it will be necessary to make a few remarks on the seat of this morbid product in general; a circumstance in the history of tubercle, which, notwithstanding its importance, and the frequent anatomical researches of which it has been the object, has not, so far as I know, been satisfactorily determined.

The prevailing opinion among pathologists is, that the seat of tuberculous matter is the cellular tissue of organs;—that it may, however, be formed on secreting surfaces, as in the mucous follicles of the intestines; perhaps in the air-cells and bronchi; the surface of the pleura and peritoneum; and likewise in false membranes, or other accidental and new products; and in the blood itself.

Seat of Tuberculous Matter.—Considered in a general point of view, and in relation to the different tissues, systems, and organs of the body, the mucous system is by far

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the most frequent seat of tuberculous matter. In whatever organ the formation of tuberculous matter takes place, the mucous system, if constituting a part of that organ, is, in general, either the exclusive seat of this morbid product, or is far more extensively affected with it than any of the other systems or tissues of the same organ. Thus the mucous system of the respiratory, digestive, biliary, urinary, and generative organs, is much more frequently the seat of tuberculous matter than any other system or tissue which enters into the composition of these organs. The coloured Plates I. and II. furnish the clearest evidence of the formation of tuberculous matter in the mucous system of all these organs. I have shewn it in the lungs, formed on the secreting surface, and collected within the air-cells and bronchi; the intestines, in the isolated and aggregated follicles; the liver, in the biliary ducts and their extremities; the kidneys, in the infundibula, pelvis, and ureters; the uterus, in the cavity of that organ, and fallopian tubes; and the testicle, in the tubuli seminiferi, epididymis, and vas deferens.

The formation and subsequent diffusion of tuberculous matter is also observed on the secreting surface of serous membranes, particularly the pleura and peritoneum; and in the numerous minute cavities of the cellular tissue. The accumulation in the lacteals and lymphatics, both before and after they unite to form their respective glands, is frequently very considerable. Striking examples of the formation of tuberculous matter in those different parts will be found in Plates III. and IV.

In Plate III. are also given representations of tuberculous matter in the substance of the brain and cerebellum, in accidental cellular tissue, and in the blood. Examples might have been given of the formation of this morbid product in a greater number of organs, and likewise in several accidental products, with which it is found occasionally combined, had the limits of the work permitted me to do so. I do not think, however, there is any reason to regret the want of these additional illustrations, as the important facts just enumerated, with regard to the *seat* of tuberculous matter, are clearly and fully demonstrated by those I have selected from the numerous collection of delineations of tubercular diseases in my possession.

External Configuration, or Form of Tuberculous Matter.—The round form which this substance is said to present is a purely accidental circumstance, is common to many other morbid products, and expresses one only, and perhaps the least important, of the many forms which this matter assumes in the several organs in which it is found. Thus, from the homogeneous nature of the cerebral substance, and the equal resistance which at every point it opposes to the accumulation of the tuberculous matter, the form of the latter must be nearly round. Such also, and for similar reasons, is its form in the cellular tissue. But in other organs the form of this morbid product is as various as that of the parts in which it is contained. It assumes the form of a shut, or open globular sac if confined to the secreting surface, and of a solid globular tumour of various sizes, if it fills completely the cavity of the air-cells; and, for similar reasons, it presents in the bronchi a tubular or cylindrical form, having a ramiform distribution, terminated by a cauliflower arrangement of the air-cells. In the mucous follicles its form is similar to that which it receives from the air-cells. In the biliary system it has a racemiform distribution, from its being contained in the ducts and their dilated bulbous extremities. In the cavity of the uterus and fallopian tubes; the infundibula, pelvis, and ureters, it is moulded to the respective forms of each of these parts; and such also, it is obvious, must be the case when it is contained in the seminiferous ducts and prostate gland, in the lacteals, lymphatics, and their glands. On the surface of serous membranes, whether natural or accidental, it may have either a globular or lamellated form, as the

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secretion in which it originates may have taken place in distinct points, or from a continuous surface of greater or less extent.

When the secretion of tuberculous matter takes place in such a manner as to become disseminated throughout a considerable extent of an organ, as when it is said to be infiltrated, it has then no definite form, unless it occupies, for example, the whole of the lobe of a lung, when it assumes that of the affected lobe. The granular arrangement of tuberculous matter in the lungs is owing to the accumulation of this morbid product in one or more contiguous air-cells; and the lobular character, which it sometimes presents in the same organ, is produced by its being confined to the air-cells of a single lobule, the neighbouring ones being healthy. Examples of these three forms are given in Plate IV.

Consistence and Colour of Tuberculous Matter.—Tuberculous matter does not acquire its maximum of consistence until an indefinite period after its formation. It is frequently found in its primitive state, in the bronchi, air-cells, biliary ducts and their dilated extremities, in the cavity of the uterus and fallopian tubes, &c. resembling a mixture of soft cheese and water, both in consistence and colour; but when much resistance is offered to its accumulation, as in the lymphatic glands, and even sometimes in the air-cells of a whole lobule, it may feel as firm as liver or pancreas. These extreme degrees of consistence of tuberculous matter depend not only on the resistance which the tissues of these and other parts oppose to its accumulation, but also on the removal of its watery part some time after it has been deposited. Hence it follows that tuberculous matter may, when first perceived, be either very soft or remarkably firm. In the first case it is pultaceous, and feels somewhat granular when rubbed between the fingers; in the second, friable; and in both it is of a pale yellow colour, and opaque.

The grey semi-transparent substance, already alluded to, by no means necessarily precedes the formation of the pale yellow or opaque tuberculous matter; it is, indeed, observed in a few only of the many organs in which the latter is found. Thus it is never seen in the cavity of the uterus or fallopian tubes; in the ureters, pelvis, or infundibula of the kidneys; in the mucous follicles of the intestines; in the lacteals or lymphatics; in the biliary ducts; nor do I recollect to have seen it in the cerebral substance. I have never met with it in the bronchi, unless in some of their most minute or terminal branches. On the contrary, the semi-transparent substance is frequently seen in the air-cells and on the free surface of serous membranes, particularly the peritoneum; and in both it is certainly sometimes observed to *precede* the formation of opaque tuberculous matter; because, first, a number of cells of the same lobule are seen filled with the former, whilst the remaining cells contain the latter substance; secondly, because on the peritoneum the grey semi-transparent substance is generally more abundant than the pale yellow opaque matter; and, thirdly, because a small nucleus of the latter is frequently inclosed in a considerable quantity of the former. The following is the explanation which I would offer of these exceptional conditions to the regular and ordinary formation of the tuberculous matter. But, first of all, it is necessary to remark that the formation and manifestation of this matter as a morbid product cannot take place unless the fluid from which it is separated—the blood—has been previously modified. This important fact being admitted for the present, it is obvious that a healthy secreting surface may separate from the blood not only the materials of its own peculiar secretion, but also those of tuberculous matter. Such is, indeed, what takes place in the air-cells. The mucous secretion of their lining membrane accumulates where

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it is formed; but it is not pure mucus; it contains a quantity of tuberculous matter mixed up with it, which after a certain time is separated, and generally appears in the form of a dull, yellow, opaque point, occupying the centre of the grey, semi-transparent, and, sometimes, inspissated mucus. This process of separation of tuberculous matter from secreted fluids is strikingly exemplified in tubercular peritonitis. When we examine the peritoneum thus affected, the three following stages of the process are frequently extremely well marked: first, on one portion of this membrane there is seen a quantity of recently secreted coagulable lymph; secondly, on another we find the same plastic semi-transparent substance, partly organized, and including within it, or surrounding a globular mass of tuberculous matter; and, lastly, on another part the coagulable lymph is found converted into a vascular or pale cellular tissue, covered by an accidental serous membrane, beneath which, and external to the peritoneal or original secreting surface, the tuberculous matter is seated, having the form of a round granular eminence, resembling in colour and consistence pale firm cheese.

In this as well as in the preceding case we cannot but perceive that the formation of tuberculous matter originates in a process similar to that of secretion; that its separation from the blood may be accompanied with that of natural and also other morbid secretions; and hence the reason why its physical characters are sometimes obscured, particularly in the first stage of its formation.

Composition of Tuberculous Matter.—The composition of this matter, when examined anatomically and chemically, presents considerable variety. I have already said that it is essentially composed of a cheesy-looking material, without any trace of organization. It has, in fact, no definite internal arrangement, and the changes of bulk, consistence, and colour which it undergoes, are entirely dependent on the influence of external agents. In some animals, but more particularly in the cow, it is frequently found to present a concentric, lamellated arrangement, which, however, does not belong to the tuberculous matter. It is owing to the presence of albumen, and sometimes even of fibrine, which, as in the cases already referred to, are secreted along with tuberculous matter. These substances, intermixed with tuberculous matter, are found lining the bronchial tubes, or filling up their entire cavity, and forming masses sometimes an inch in diameter; they often assume the form of globular membranous cysts when they are contained in the air-cells, and then have a striking resemblance to hydatids; or, lastly, they present the form of detached tubes or globular membranes, rolled up and mixed with tuberculous matter, like layers of boiled albumen or dead hydatids; a circumstance which has been taken advantage of to support the theory of the hydatid origin of tubercles;—a theory which, if not founded in error, must obviously be regarded as extremely limited in its application, since I have shewn that tuberculous matter is in general formed *ab origine* on the secreting surface of hollow organs, where it is seen as distinctly as if it had been thrown into them from a syringe.

The chemical composition of tuberculous matter varies not only at the different periods at which it is examined, but also in different animals, and, probably, in different organs. In man it is chiefly composed of albumen with various proportions of gelatine and fibrine; in the cow, in particular, it contains a large proportion of the earthy salts, in which the phosphate of lime is said either to predominate, or to exist in the same proportion, along with the carbonate of the same earth, as it does in bones.

The most important fact connected with the chemical composition of tuberculous matter is, that, either from the nature of its constituent parts, the mode in which they are combined, or the conditions in which they are placed, they are not susceptible of

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organization, and consequently give rise to a morbid compound, capable, as I have already said, of undergoing no change that is not induced in it by the influence of external agents.

Softening of Tuberculous Matter.—When the process of softening takes place in tuberculous matter, it is clear, from what I have just stated, that it cannot be owing to any change originating in this morbid product. Besides, when speaking of the *consistence* of this matter, I showed that in many organs it is always in a state of fluidity, and, consequently, does not require to undergo the change in question, supposed to be necessary to, or at least to facilitate, its expulsion. When the tuberculous matter has become firm, owing to the circumstances which I have already explained, it may at some future period be converted into a granular-looking pulp, or pale grumous fluid of various colours, from the admixture of serosity, pus, blood, &c., which have been effused or secreted by the tissues subjected to its irritating influence. The pus and serosity pervade the substance of the tuberculous matter, loosen and detach it. These changes are further promoted by atrophy, ulceration, or mortification of the surrounding or enclosed tissues, the bloodvessels of which have been compressed or obliterated by the tuberculous matter.

If these changes take place slowly, as in the lungs, the tuberculous matter is expectorated in the form of a grey, somewhat puriform-looking fluid; but if they are effected speedily, it is often detached and expelled in masses of various sizes, resembling fragments of cheese which have been left some time in water.

The process of softening of tuberculous matter is said always to commence in the centre, not only of masses of this substance, but likewise of every individual portion of it which has assumed the round or tubercular form. This opinion is extremely incorrect; and, indeed, the explanation which I have just given of the process shows that such cannot be the case. However, there must be some real or apparent circumstance connected with this *central* softening, so minutely described by Laennec, which has not been understood by this ingenious author, nor by those who disagree with him on this particular point. The description which I have given of the formation, seat, and forms of the tuberculous matter, enables me to offer a satisfactory explanation of the appearances which have led Laennec and others into error, regarding the commencement of softening of this substance.

It has been already stated that when tuberculous matter is formed in the lungs, it is generally contained in the air-cells and bronchi. If, therefore, this morbid product is confined to the surface of either, or has accumulated to such a degree as to leave only a limited central portion of their cavities unoccupied, it is obvious, that when they are divided transversely, the following appearances will be observed:—1. a bronchial tube will resemble a tubercle having a central depression or soft central point, because of the centre of the tube not being, or never having been, occupied by tuberculous matter, and because of its containing a small quantity of mucus or other secreted fluids;—2. the air-cells will exhibit a number of similar appearances, or rings of tuberculous matter grouped together, and containing in their centre a quantity of similar fluids.

When the bronchi or air-cells are completely filled, the tuberculous matter presents no such appearances as I have described; and hence the reason why tubercle, in such circumstances, has been said to be still in the state of *crudity*, or that condition which precedes the softening process.

Softening begins most frequently at the circumference of firm tuberculous matter, or

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where its presence as a foreign body is most felt by the surrounding tissues. Hence the reason why softening is frequently seen making its appearance in several points of an agglomerated mass of this substance, which has included within it portions of the tissues in which it was formed. This is frequently observed in the lungs, and cellular tissue in other parts; whereas in the brain, the substance of which has from the commencement been separated and pushed outwards by the tuberculous matter, the softening process begins, and is always most marked, on the circumference of this morbid product.

I have already alluded to the formation of the opaque, central point, described by Laennec, and which has been shewn to originate in the separation of the tuberculous matter from the other secretions which sometimes accompany its deposition.

There is another circumstance which should have been noticed before, and which requires some explanation—viz. that state of tuberculous matter which is said to be *encysted*. Encysted tubercle has generally been described as existing in the lungs, but I feel perfectly satisfied that the term encysted, whether applied to pulmonary tubercle or to tubercle in any other organ, is almost always incorrect. In the lungs encysted tubercle is a deception, the distended walls of the air-cells having, in all probability, in almost every case been taken for cysts. In like manner the dilated bulbous extremities of the biliary system have been described as cysts of the liver containing tuberculous matter; and I have already said that the dilated air-cells, particularly in the cow, which vary from the size of a pea to that of a cherry, have frequently been regarded as hydatids.

We do, however, meet with encysted tuberculous matter, but not until it has undergone important changes which precede its ultimate removal from the organ in which it was formed: I shall explain these and other changes presently.

Progress and Termination of Tuberculous Matter.—I have already noticed several of the changes which take place in the tissues which are the seat, or are in the immediate vicinity, of tuberculous matter, as well as those which are observed to occur in this morbid product itself. Mechanical and inflammatory congestion; softening and induration; atrophy, ulceration, and mortification, are morbid states which may be produced either directly or indirectly by this substance; but as these changes constitute distinct subjects, they will afterwards be treated under their respective heads. I shall, therefore, confine my remarks, for the present, to such anatomical facts as seem to me to demonstrate the entire removal of tuberculous matter from an organ, or, in other words, the cure of tubercular diseases.

Every physician must believe in the cure of scrofulous swellings, even without ulceration or suppuration having taken place in them. Such cases, I am aware, are regarded by some as simple, chronic, inflammatory swellings of the lymphatic glands; but this opinion I by no means conceive to be correct, for among the great number of cases which I have examined I have never found these glands, when generally affected, exempt from the presence of tuberculous matter; and even when the cutis is pale (if they are situated under this tissue), I have sometimes found them almost completely filled with this morbid product. When, therefore, enlarged glands, in a scrofulous patient, ultimately disappear, we may conclude, almost with certainty, that we have witnessed the cure of a tubercular disease.

Tabes mesenterica has been known to terminate favourably. I had an opportunity of examining, in a case of this kind, the mesenteric glands, and thereby of determining the certainty of the cure. The patient, who, when a child, was affected with this disease,

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and also swellings of the cervical glands, some of which ulcerated, died, at the age of 21, of metritis, the seventh day after delivery. Several of the mesenteric glands contained a dry, cheesy matter, mixed with a chalky-looking substance; others were composed of a firm cretaceous substance; and a tumour, as large as a hen's egg, included within the folds of the peritoneum, and which appeared to be the remains of a large agglomerated mass of glands, was filled with a substance resembling a mixture of putty and dried mortar, moistened with a small quantity of turbid serosity. In the neck, and immediately beneath an old cicatrix in the skin, there were two glands which contained in several points of their substance (which was healthy) small masses of hard cretaceous matter.

I have also been able to trace the several steps of the same curative process in the bronchial glands, in individuals who had recovered from scrofula and pulmonary phthisis, but who died some time after of other diseases. I have found these glands situated at the bifurcation of the trachea, where they are generally most frequently and most extensively affected, as well as some way up the trachea, containing a greater or less quantity of a substance resembling putty or dry mortar, the consistence of which was sometimes equal to that of sandstone or bone. This substance has generally a stellated form, or presents a number of sharp spiculæ projecting from a central mass, which excite inflammation, ulceration, and hence perforation of the walls of the trachea or bronchial tubes with which they come in contact. A direct communication is thus formed between the cavity of the air-tubes and the diseased glands, through which the cretaceous bodies pass; and they are rejected along with the expectorated fluids. I have seen several examples of cure of tubercular disease of the bronchial glands, effected in the manner just described. The patients were generally advanced in years, and had frequently observed the cretaceous matter in their sputa, portions of which have been shown to me, and were found to present all the physical characters of that which was afterwards detected in the bronchial glands.

When these glands have evacuated the whole of their contents, they are found atrophied, and converted into a fibrous tissue, which fills up the external orifice of the perforated air-tube. The accidental opening now contracts, becomes obliterated, and leaves in its place a puckered depression or cicatrix, seen on the internal surface of the air-tube.

Similar appearances indicating the removal of the serous and albuminous parts of the tuberculous matter, and the condensation of its earthy salts, have frequently been observed in the lungs of persons whose history left no doubt as to their having, at some period of their lives, been affected with tubercular phthisis. The important fact of the curability of this disease has, in my opinion, been already established by Laennec. I shall therefore only shortly allude to those changes which take place in the tuberculous matter, pulmonary tissue, and bronchi, which indicate that this fortunate termination of phthisis has taken place. The tuberculous matter, whether contained in a bronchial tube, the air-cells, or cellular tissue of the lungs, has assumed a dry, putty-looking, chalky, or cretaceous character. If these changes are observed in an excavation, the surrounding pulmonary substance is generally dark-coloured; and if the excavation exists in the course of large bronchial tubes, those situated between the excavation and the periphery of the lungs are obliterated, whilst those in the opposite direction terminate either in a shut extremity near the excavation, or are continuous with the lining membrane or accidental tissue which encloses the altered tuberculous matter. The existence of this accidental tissue is an important circumstance as regards the cure of tubercular excavations. It is formed by the effusion of coagulable lymph on the surface of the excavation, or in the substance of the contiguous pulmonary tissue; has at first, and so long as a

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ready exit is afforded to its secretion, the characters of simple mucous tissue ; but at a later period, and especially when the latter condition is wanting, it becomes gradually and successively converted into serous, fibrous, fibro-cartilaginous, and cartilaginous tissues.

The cartilaginous and the osseous transformations of this accidental tissue are, however, rare, particularly the latter. It much more frequently retains the fibrous character, and possesses the property of contracting so as to diminish the bulk of the excavation, and carry with it the pulmonary tissue with which it is connected. The diminution of bulk which accompanies the removal of the tuberculous matter, and the contraction of this accidental tissue, give rise to a puckering of the lung, which is best seen where the pleura is forced to follow the retrocession of the pulmonary substance beneath it, and around what is called the cicatrix : for there sometimes remains only a small globular, oval, or even linear portion of fibrous or fibro-cartilaginous tissue, in a part of the lung, where, from the extensive puckering around it, there must have formerly existed an excavation of considerable extent.

When the tuberculous matter is contained within the bronchi, or a cavity formed by the dilatation of the air-cells, it does not appear that any accidental tissue is formed during the progress of the cure. The matter is gradually removed by expectoration, if the bronchi remain pervious, or by absorption if they become closed ; and then we have the same obliteration of the terminal branches already alluded to, and the same puckering of the surrounding tissues : all these appearances have been represented in Plate IV., and will be better understood when pointed out in the figures.

The cure of tubercular diseases in other organs has not been satisfactorily demonstrated. I have, however, as was before done by Jenner and since by Dr. Baron, frequently produced tubercles in the liver of the rabbit, and have afterwards followed their complete removal by absorption and by excretion. When this is accomplished by the latter process, which is most commonly the case, no trace of the disease remains ; but when effected by absorption, I have often found the surface of the liver marked by irregular furrows or depressions, apparently produced by atrophy of the substance of the organ around the seat of the tuberculous matter.

Such is a general description of the seat, form, consistence, colour, and composition of tuberculous matter ; of the successive changes which it undergoes during the several periods of its existence, as well as of some of the more remarkable changes which take place in the organs in which it is formed, and more particularly of those which are generally regarded as evidence of its entire removal, and thereby demonstrate the curability of tubercular diseases.

The comparative frequency of these diseases in males and females, in different animals, at different periods of life, and in different organs of the same or of different individuals, are subjects which, it may be said, belong essentially to the province of pathological anatomy : but even a short sketch of the numerous facts which pathologists have been able to collect in regard to several of these subjects, would be incompatible with the limits of this work ; in which, moreover, the *elementary morbid anatomy* of diseases is the exclusive subject of investigation. We propose, however, to give due consideration to this department of pathological anatomy in the *Elements*, where we shall also treat of the formation of tuberculous matter considered in relation to those conditions of the economy, hereditary or acquired, in which it appears to originate ; of the influence of disease of particular organs in its production ; of the modifications of function to which its presence gives rise ; and of the means which the knowledge thus derived suggests as most likely to prevent its formation, or facilitate its removal.

DESCRIPTION OF THE PLATES.

PLATE I.

Fig. 1. Represents a section of the greater part of the superior lobe of the left lung. A, left division of the trachea and three large bronchi laid open; one of these bronchi is filled with a mass of tuberculous matter, B, a portion of which projects into the cavity of the tube, and from thence is seen extending into the smaller branches and air-cells, the latter presenting a granular aspect and cauliflower arrangement. The ramiform and cauliflower arrangement of the tuberculous matter is still more conspicuous at C. The walls of the bronchi and air-cells are seen entire, and including within them this morbid product, a cylindrical portion of which projects into a vomica formed in the continuity of the principal air-tube. Several very small lateral bronchi are shewn at D, with their air-cells filled with tuberculous matter, and the same substance projecting into the cavities with which they communicate. Masses of tuberculous matter of various sizes and forms are shewn scattered throughout the substance of the lung. Some of them, E, are represented divided, and present a nearly uniform aspect, resembling the cut surface of firm cheese; others, F, are shewn entire,—that is to say, the pulmonary tissue has been removed from around them, to shew that they were formed of dilated air-cells filled with tuberculous matter.

Two forms of vomicae are seen at G and H; one of them in the situation, the other at the extremity, of a large bronchus. Both of them contained soft and firm tuberculous matter; their internal or mucous surface was pale, and covered with a layer of the same substance. The mucous membrane of the large bronchial tubes was of a livid red colour, and in the principal branch, K, it was ulcerated, and here and there in a state of sphacelus.

Fig. 2 is a beautiful specimen of the disposition of the tuberculous matter described above. A number of irregular anfractuous cavities, of various sizes, marked A, are represented lined with a layer of tuberculous matter, B, of a greyish green colour internally, and of a pale yellow colour externally. On raising this substance, prolongations of it were seen passing either into smaller cavities, or into minute lateral and terminal bronchi, and from these into the air-cells. The bronchi and air-cells are represented at D and E; both of them were dilated, some of the former varicose, the latter forming small cauliflower-shaped expansions. The tuberculous matter is seen in the substance of the lungs, which was injected and œdematous, under various forms, according as it is seated in the air-cells or bronchi, or to the manner in which either of these are exposed, or the quantity which they contain of this substance.

Fig. 3, a section of a small portion of the upper lobe of the lung, the substance of which is studded with small granular-looking masses of tuberculous matter, formed by the re-union of the air-cells either partially filled, or distended with this substance. Some of them, A, are represented entire; others, B, divided. The former have one or more central depressions, from the tuberculous matter not occupying the central portion of the air-cells; the latter have a smooth surface and no central opening, from the same substance filling the whole cavity of the cells, and from its being covered by the walls of these cavities. This arrangement of the tuberculous matter is better seen at C, in a bronchial tube and its corresponding air-cells, which have been exposed. Three vomicae are represented in the same portion of lung; two of them very small, the third much larger, formed by the separation and expulsion of the tuberculous matter.

There is also seen in this figure a small quantity of the black pulmonary matter

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which so frequently accompanies the presence of tubercle. It does not exist in the tuberculous matter, but in the cellular tissue or capsules of the air-cells, as indicated at F.

Fig. 4 represents a portion of the lung of the cow, which may be said to present a panoramic view of the seat of tuberculous matter, and the forms which it assumes in the human species, as shewn in the preceding figures. A, a large bronchial tube laid open, which was distended with tuberculous matter. A portion of this tube is represented empty, in order to shew the tuberculous matter B, passing into the smaller bronchi and air-cells C, which are seen covered by their proper tunics, dilated, and grouped together along the course of the principal tube, in the form of a chain of globular tumours of various sizes.

A similar state of the air-cells is seen at D and E, where they terminate under the pleura. At D they are dilated into the form of a pyriform tumour of considerable size, from which the tuberculous matter is seen projecting into the corresponding bronchial tube. At E they form globular tumours of various sizes, and also a ramiform disposition, being seen connected with their bronchi, which are but partially dilated.

Several dilated air-cells are represented at F, empty, in order to shew the manner in which they are disposed of to form tumours, similar, for example, to the one marked D. The dilatation of a single group of air-cells forms frequently a perfectly smooth globular tumour, resembling an hydatid, as at G; and which, when cut across, as at H, is found to consist either of a transparent or opaque membrane, filled with tuberculous matter. Groups of dilated air-cells and bronchi, K, varying in size and form, are represented scattered throughout the substance of the lungs. A large bronchus is shewn at L, greatly dilated, having the form of a chain of contiguous lateral swellings, and communicating with each other and with the parent tube, as indicated by the probe M. Several tumours, N, are represented attached to the pleura pulmonalis, either by a broad base or by slender necks. They were formed of a serous capsule, containing tuberculous matter similar to that formed in the bronchi. The pleura costalis and omentum, in this case, were studded with similar tumours, some of them, in the latter, as large as an orange. The gall-ducts, too, contained a great quantity of tuberculous matter; and some of them, the natural size of which does not exceed a crow-quill, were so dilated by the accumulation of this substance within them, that they nearly equalled in diameter the human gall-bladder.

PLATE II.

Fig. 1. This figure affords a striking example of the formation of tuberculous matter in the cavity of the uterus and fallopian tubes, as well as ulceration of the follicles and mucous membrane of the vagina.

A. Cavity of uterus laid open, and nearly filled with masses of cheesy-looking tuberculous matter. The walls of the uterus, thicker and more vascular than in the healthy state, contain two or three small masses, B, of the same substance. Both fallopian tubes, C, are dilated; the left, completely filled with soft tuberculous matter, and laid open towards its inferior extremity, that this substance may be seen. The right tube was filled with a turbid, milky-looking fluid. The internal surface of the vagina, D, presents a great number of ulcers, similar to those so frequently met with in the trachea of patients who die in the last stage of phthisis. The ulcers were apparently formed in the follicular structure of the vagina; some of the follicles,

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enlarged and presenting a central opening, are distinctly seen in the figure. The form of the ulcers is round, oval, or irregular, none of them larger than a split pea; their edges sharp and pale; and their bottoms either pale or slightly vascular.

Fig. 2 represents a fallopian tube separated from the uterus of another patient, who, like the former, died of phthisis. It is dilated, tortuous, obliterated at its inferior extremity, and filled with firm tuberculous matter, D. It was perforated, and adhered to the uterus at the point E.

Fig. 3 represents sections of different portions of the same tube, D, the tuberculous matter, surrounded by the walls, A B C, of the tube.

Fig. 4 is a representation of one of the testes of a young man who died of phthisis. The body of the testicle, A, contained a multitude of pale yellow-coloured granular bodies of various sizes, which were obviously formed by the accumulation of tuberculous matter in the tubuli seminiferi. The epididymis was as thick as the little finger, and its convoluted ducts were obviously filled with tuberculous matter; for on pressing the epididymis, this substance flowed not only from the divided extremities of the ducts, B, but also at the cut extremity, C, of the vas deferens.

Fig. 5, a longitudinal section of the right kidney of a highly scrofulous girl. All the cavities of this organ were completely filled with tuberculous matter, the tubular portion destroyed, and the cortical greatly atrophied. The kidney is represented after the removal of the greater part of the tuberculous matter. Three of the infundibula are seen, at A, filled with this matter, which passes into the pelvis B, and from thence into the ureter C. A tube is seen passed from one of the infundibula through the ureter, from the cut orifice of which projects a tubular layer of the same substance. The destruction of the tubulous portion of the kidney is indicated by the form and arrangement of the cavities, E; and the atrophy of the cortical substance, F, is conspicuous all round the cut margin of the kidney.

Fig. 6 furnishes a beautiful illustration of the seat of tuberculous matter in the liver of the rabbit. A portion of the liver is represented entire, that the usual form, A, of the tuberculous matter in the substance of this organ, beneath the peritoneum, may be seen. B represents similar collections of the same morbid product, which, from the substance of the liver having been carefully dissected away from around a portion of the biliary system, is seen lodged in the extremities of the ducts, which are dilated into the shape of pyriform sacs of various sizes. In these sacs, as well as in the biliary ducts, the tuberculous matter was of the colour and consistence of cream, and was made to flow out at the cut extremity, C, of the ductus communis choledochus, when pressure was applied either to the hepatic ducts or to their bulbous extremities. The pyriform sacs presented, externally, a smooth uniform surface, except in two or three instances, in which they were slightly lobulated, as at D. Internally, some of them had a cellular aspect, but the most of them were uniformly hollow. The fundus, E, of several was connected with the substance of the liver by what appeared to be small bloodvessels. F, the gall-bladder distended with bile.

PLATE III.

Fig. 1 represents a portion of the ileum laid open and spread out, to which are attached a piece of the mesentery, several of the mesenteric glands and lacteals. The tuberculous matter is shewn occupying the mucous follicles of an enlarged aggregated

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gland, A. It is also seen projecting from the orifices of the solitary glands or follicles, B. At C these follicles are represented in a less advanced stage, their central orifices just beginning to be visible, from the distention occasioned by the tuberculous matter collected within their cavities. The first stage of this diseased state of these follicles is seen on various points of the surface of the mucous membrane, and is indicated by the presence of a small, round, somewhat conical, elevation of a light grey or pale straw colour. Ulceration of the glandulæ aggregatæ and solitariae, and of the mucous, cellular, and muscular coats of the intestine, is represented at D, E, F, as the consequence of the presence of tuberculous matter in these tissues. D, three small oval ulcers, with sharp, smooth, pale borders, formed in the mucous membrane, in the situation of the isolated follicles, their bottoms consisting of the submucous cellular tissue. Ulceration of the aggregated follicles, mucous, sub-mucous, muscular, and sub-peritoneal tissues, is represented at E and F. E, the muscular coat laid bare and ulcerated, and containing tuberculous matter in its inter-cellular tissue. F, the cellular tissue beneath the former, studded with minute round portions of the same substance.

G, the lacteals, dilated and filled with tuberculous matter, passing from the intestine into, and out of, the mesenteric glands, H, K, L, many of which are enlarged. All of them contain a greater or less quantity of tuberculous matter; one of them, L, is completely filled with it; two others, H and K, contain it in less quantity, collected either towards their centre or circumference, the unoccupied portions of both being in a state of congestion. The lacteals are represented, at M, arising from the ulcerated follicular gland, and passing beneath the mucous membrane to the mesentery; and at O they are seen forming an irregular net-work beneath the peritoneum, over the situation of an ulcer of the intestine, whence they pass to the mesentery, gradually increasing in size as they advance. One of the branches, P, was injected with mercury, the progress of which was soon arrested by the tuberculous matter, N, accumulating in front of the metal.

Fig. 2 and 3 represent tuberculous matter in the brain and cerebellum. In the former are seen three round masses of this substance, the largest, A and B, situated in the medullary, the smallest, C, in the cortical substance of the anterior lobe of the left hemisphere. The tuberculous matter has a uniform smooth aspect, of the colour and consistence of new cheese. In the latter figure this substance is seen forming a lobulated tumour, A, as large as a hen's egg, situated in the right lobe of the cerebellum, and projecting above its surface. Its consistence is cheesy, of a yellowish grey or greenish yellow colour, C, and presents here and there anfractuous cavities, containing a grumous milky-looking fluid. In the first of these cases the cerebral substance around the tuberculous matter was perfectly healthy; in the second, the medullary and cortical substances were soft where they were pressed by the tumour. The pia and dura mater, D, adhered together over the most prominent point of the tumour.

Fig. 4 represents the formation of tubercles in false membranes, covering a portion of the small intestines. This portion of intestine is covered by an accidental serous membrane, beneath which, and over the surface of the peritoneal coat, is a layer of accidental cellular tissue. The accidental serous membrane A is cut, and reflected downwards, to shew the tubercles in the cellular tissue, B, beneath it. Towards the concave border of the intestine the accidental cellular tissue, B, is separated and turned upwards; portions of the peritoneum being carried along with it, and thus exposing the circular fibres, C, of the muscular coat; between which, as well as in the accidental cellular tissue, a number of very small tubercles are distinctly seen. Larger tubercles, D,

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are seen on the surface of the mesentery, but lying beneath the accidental serous membrane.

Fig. 5 is a section of the spleen, to shew the formation of the tuberculous matter in the blood contained in the cells of this organ. The following are the changes which take place in the blood during the formation of tubercles in the spleen. In one point of the organ the blood is coagulated, firm, and dark; in another, it has lost its deep red colour, and resembles fibrine tinged with blood. In this state it has assumed a definite or circumscribed form, appearing either like small granular bodies or irregular masses of considerable size. Lastly, the red fibrine disappears, and is replaced by a nearly colourless substance, composed of pale fibrine and tuberculous matter. The first of these successive changes is seen at A, and also around a mass of fibrine, B. C indicates the second change or separation of the fibrine of the blood, its round form and red colour. The separation or formation of the tuberculous matter is shown at D, in the cells of the spleen, and at B, in a large mass of fibrine. The separation of the tuberculous matter from the blood can be seen taking place in a single cell of the spleen. The tuberculous matter occupies the centre of the cell, and is surrounded by a globular layer of fibrine of considerable thickness. If the latter becomes organised, the tuberculous matter is found encysted. *Fig. 6* is a magnified view of the tuberculous matter contained in a capsule of organised fibrine.

PLATE IV.

Fig. 1. A. Solidification of a large portion of lung from infiltration of tuberculous matter, which in this case terminated in gangrene, B, in consequence of compression and obliteration of the bloodvessels C. D, obliterated bronchi.

Fig. 2. A. Vesicular deposition of grey, semi-transparent tuberculous matter in a number of contiguous lobules of the inferior lobe of the right lung, complicated with pneumonia. B, isolated miliary tubercles in the same inflamed lobe. C, agglomerated masses of pale yellow opaque tuberculous matter situated in the superior lobe, and produced by the accumulation of this matter in almost all the cells of each lobule, the surrounding tissues being healthy.

Fig. 3. Vesicular deposition of grey, demi-transparent tuberculous matter, A, in all the air-cells of a number of contiguous lobules. Several of these lobules, B, are seen on the external surface of the lung, some of them enlarged, others of their natural size, and projecting forwards, because of the healthy pulmonary tissue around them being collapsed. In both of these situations the vesicular character of the tuberculous matter is beautifully seen. The upper half of both lungs was in the same state; and owing to the density of the tuberculous matter, a great many of the pulmonary veins, C, belonging to the affected lobules, were either much compressed or obliterated. Such an extensive and complete obstacle to the pulmonary circulation was followed by effusion of blood into the bronchi, D, and frequent expectoration of more or less of this fluid. We regard this case as explanatory of the nature and origin of the hæmoptysis which so frequently announces the existence of tubercular phthisis, as hemorrhage from the lungs at this early period of the disease cannot be the consequence of perforation of the bloodvessels, a lesion which does not occur often, and generally not till towards the middle or termination of the last period.

Fig. 4 represents the changes observed to take place in tuberculous matter, exca-

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vations, bronchi, and pulmonary tissue, during the cure of phthisis. A, the remains of a large cavity formed by the dilatation of a bronchial tube of considerable size, its terminal branches and air-cells. It was situated under the pleura, had the form of a shut sac, from the bronchus, B, with which it was continuous, being obliterated. Its walls were composed of a fibrous tissue, lined with a smooth pale membrane. It contained a cheesy-looking matter, part of which, from the surface of the lung, C, being puckered, and considerably depressed in the direction of the centre of the excavation, had evidently been removed by absorption. Similar but more complete changes are seen to have been effected in the direction of the obliterated bronchus, D. Several irregular masses, E, of tuberculous matter converted into a substance like putty, occupy a portion of the pulmonary tissue in this situation; and a bit of fine wire has been passed into this substance, through a narrow canal opening into the infundibuliform extremity of the same bronchus, at which point is also seen a little of the altered tuberculous matter, which was forced out by gentle and repeated pressure, in order to shew that it was originally formed in a dilated air-tube, or in an excavation which communicated with the bronchus, D. At F is a round body, about half an inch in diameter, which indicates very distinctly a more advanced stage of the cure, probably of an excavation. It is composed of two concentric layers, the outer one dense, and of a grey colour, resembling cartilage; the inner fibrous, and enclosing a nucleus of cretaceous matter. Nearer the surface of the lung, the only remains of the tuberculous matter and the excavation in which it was originally contained, is pointed out by the presence of a small elliptical portion of fibro-cartilaginous tissue, G, in which are seen traces of its having been, like the former body, composed of concentric layers. The pulmonary tissue in which these changes are observed appears puckered, retracted, and marked with black points and streaks.

In another portion of the same lung there is seen an oval mass, H, of dry tuberculous matter, about the size of a cherry-stone. It compresses a bronchial tube of considerable size, which, towards the surface of the lung, and about an inch beyond the compressed point, is dilated, filled with tuberculous matter, and terminates in two shut prolongations, from each of which the terminal branches K are seen ramifying, obliterated, contracted, and converted into pale fibrous cords. A similar state of the terminal branches of another bronchial tube is seen at L. The obliteration of both may have been the consequence of compression produced by the cavities in their immediate vicinity, although it is also observed to follow the cure of phthisis depending on the presence of tuberculous matter in the bronchi and air-cells.

In the same figure we have an interesting view of the process by means of which the cretaceous remains of tuberculous matter are removed from the bronchial glands. One of these glands, M, situated at the bifurcation of the trachea, is represented laid open, and filled with a number of irregular spiculated bodies, composed of this matter. One of these bodies is seen making its way through a perforation, N, of the left division of the trachea, situated immediately over the diseased glands. A cicatrix of a similar perforation of the trachea is seen at O. The remains of the gland were found adhering to the external surface of the trachea, in the situation of the cicatrix.

Fig. 5 represents the state of the mesenteric glands in the case of cure of *tabes mesenterica*, to which I have alluded. A, a large tumour, the remains of a group of diseased mesenteric glands, laid open, to shew the nature of its contents, which resembled a mixture of putty and mortar. B, two of the mesenteric glands still enlarged, and containing a cheesy or wet chalky-looking substance. C, another of the same glands contracted and composed entirely of cretaceous matter. D, a healthy mesenteric gland.

Plate 1.

Fig. 1.



Fig. 2.

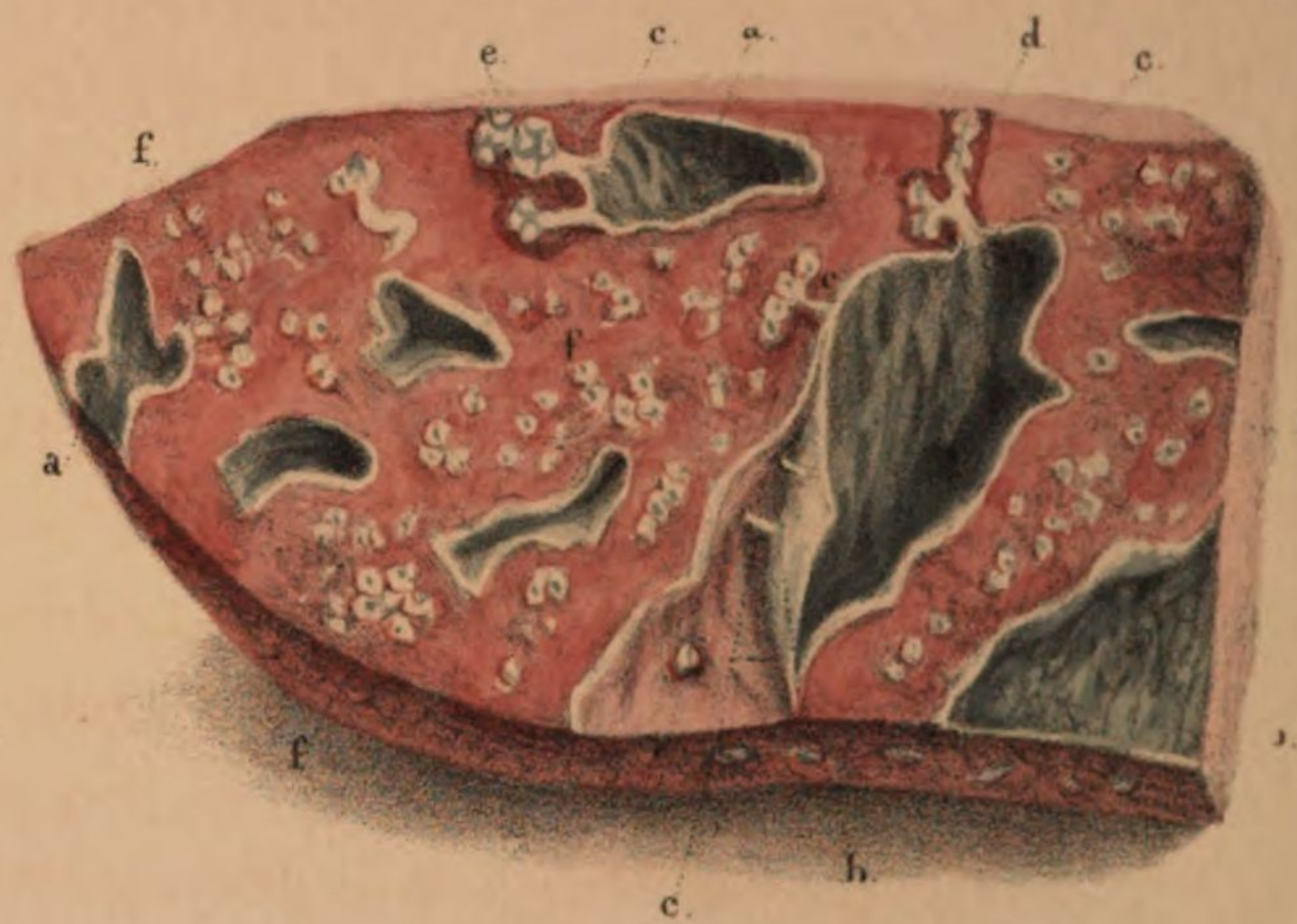


Fig. 3.

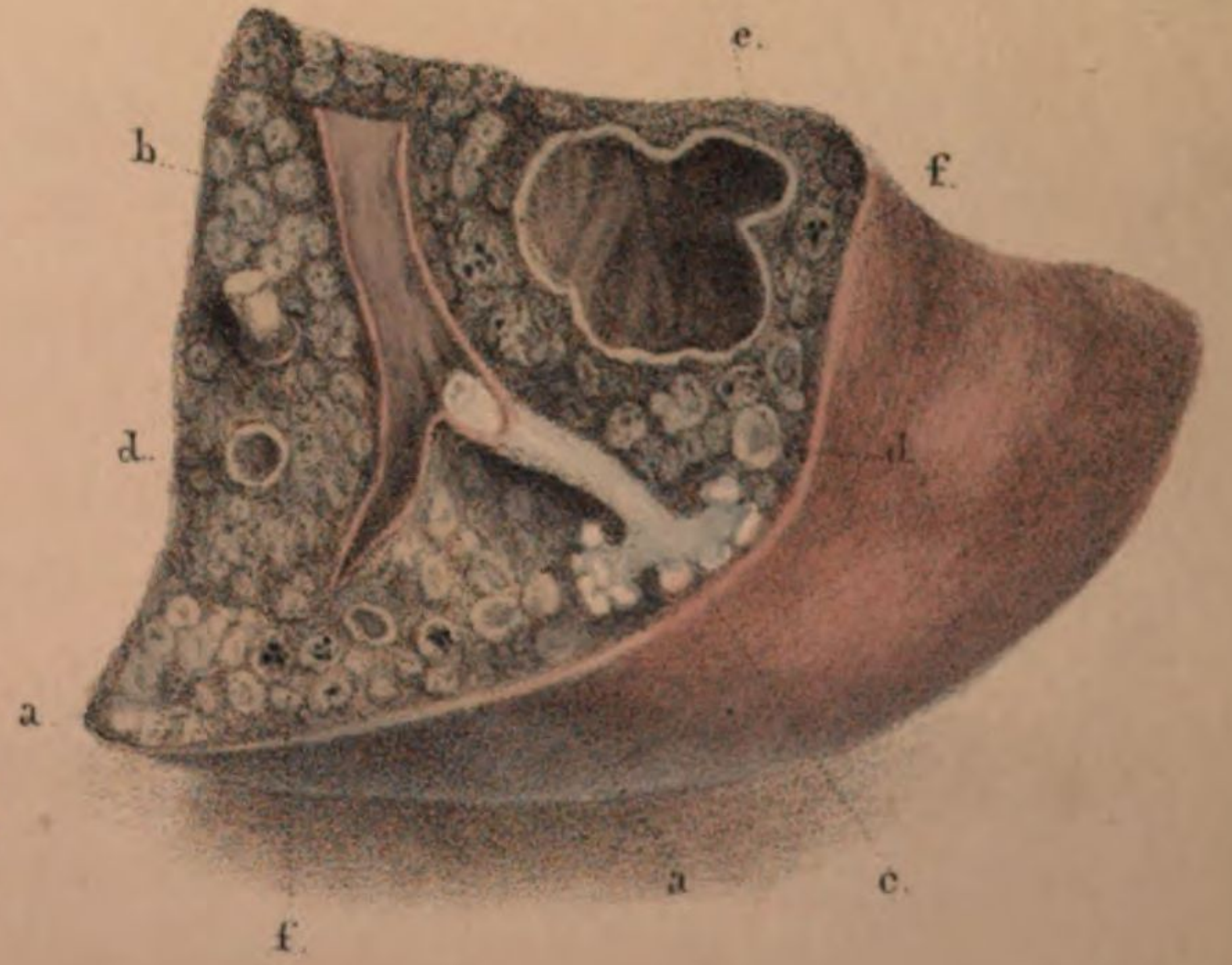


Fig. 4.

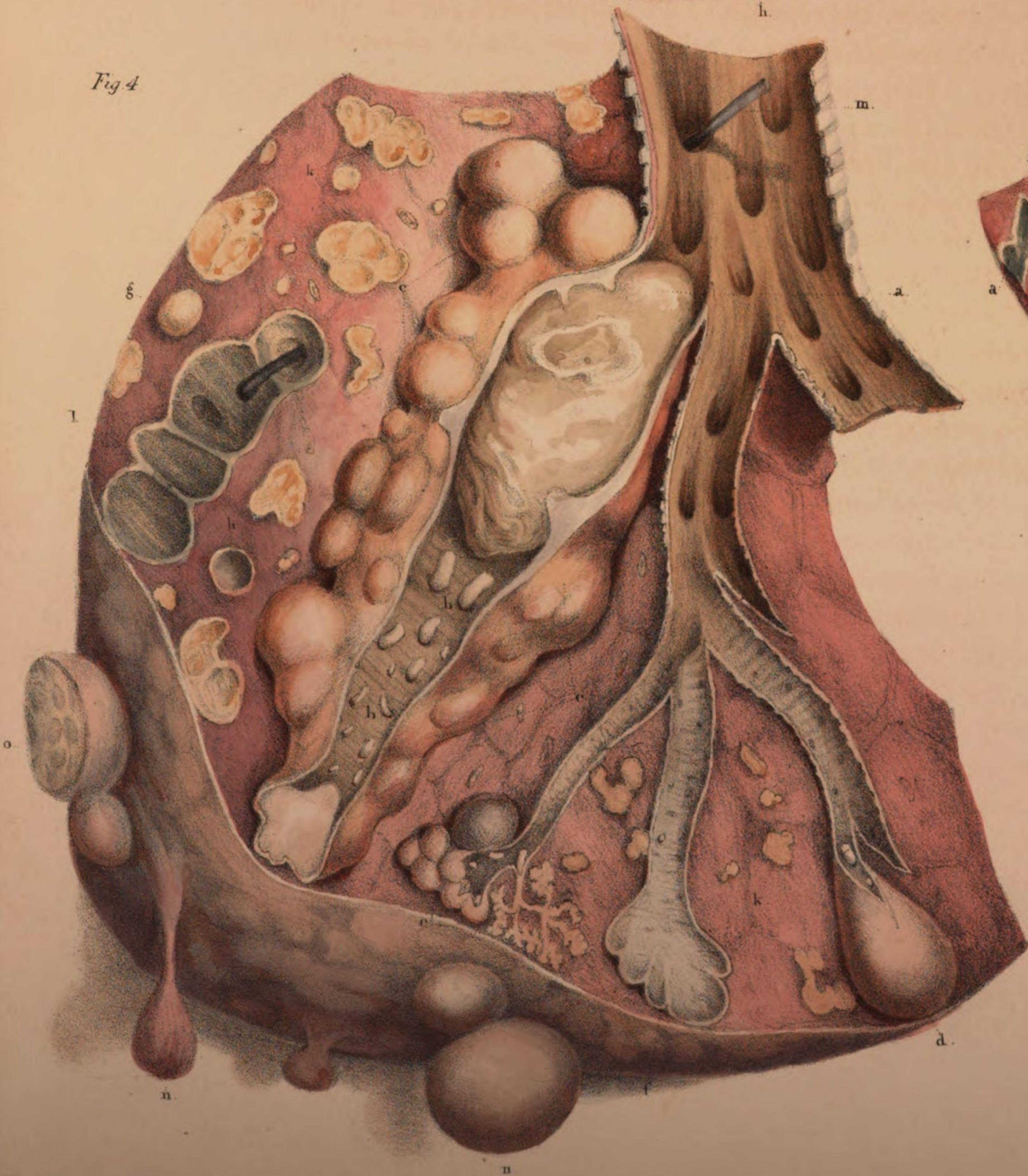


Plate II.

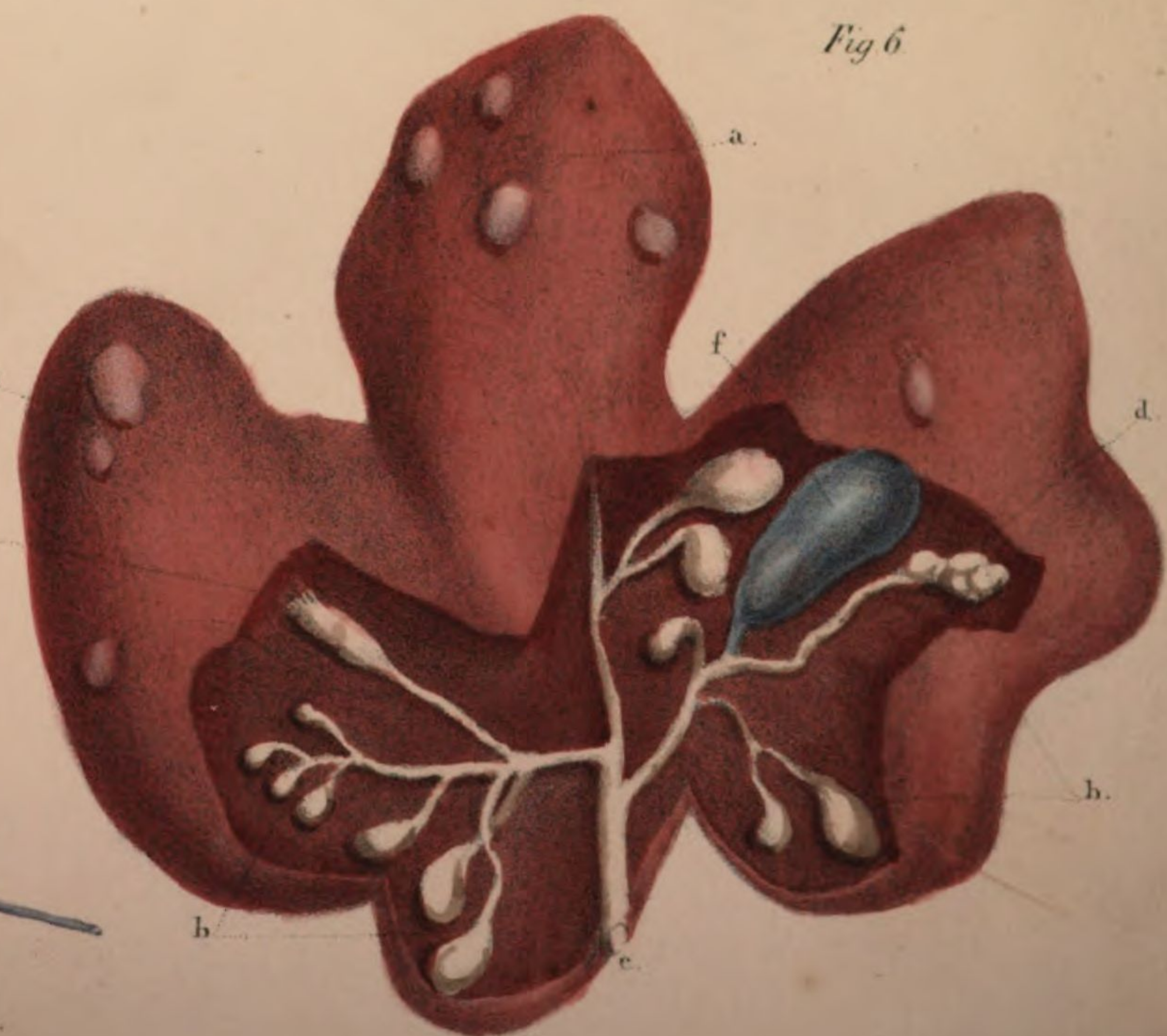
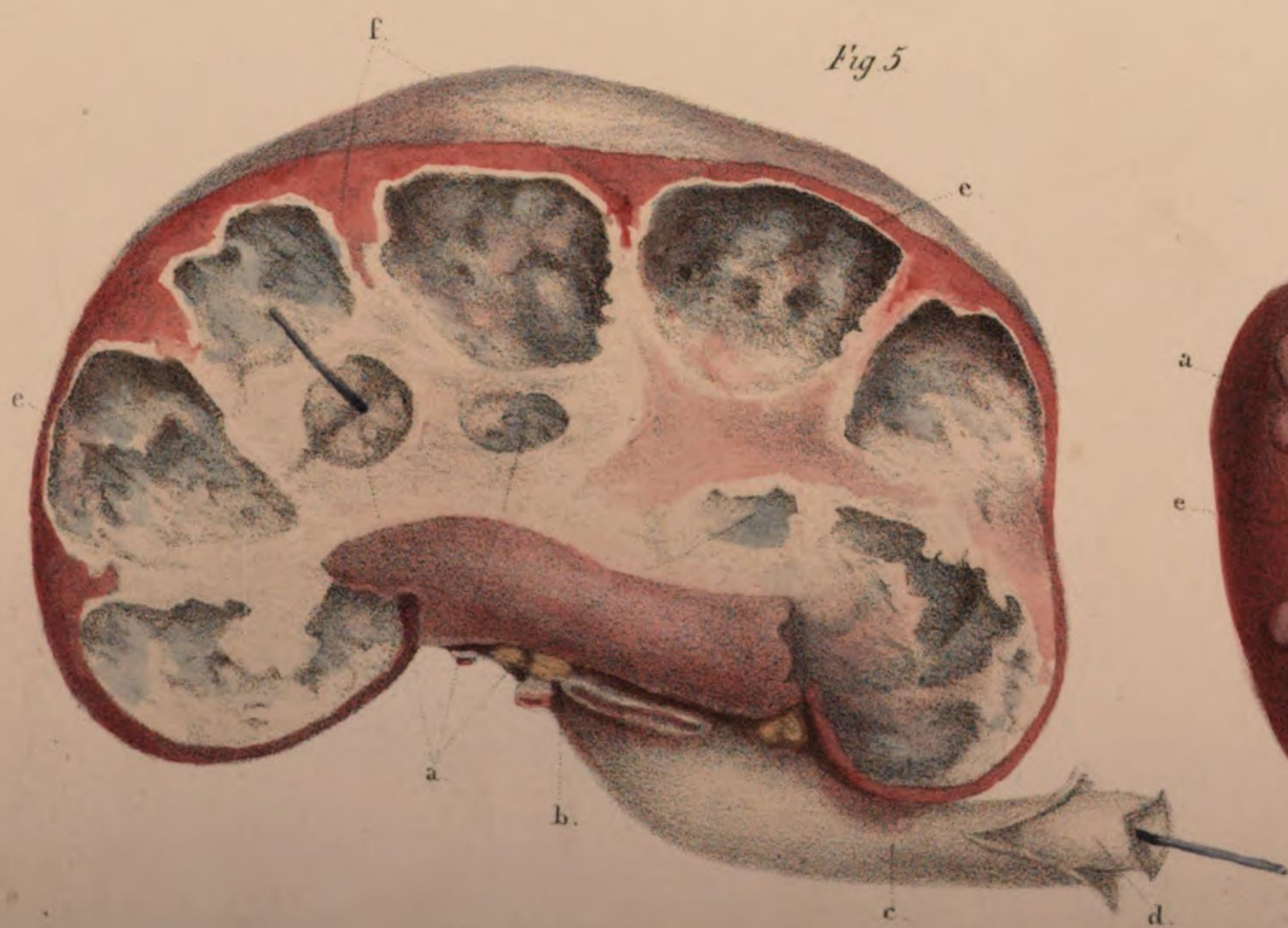


Plate III.

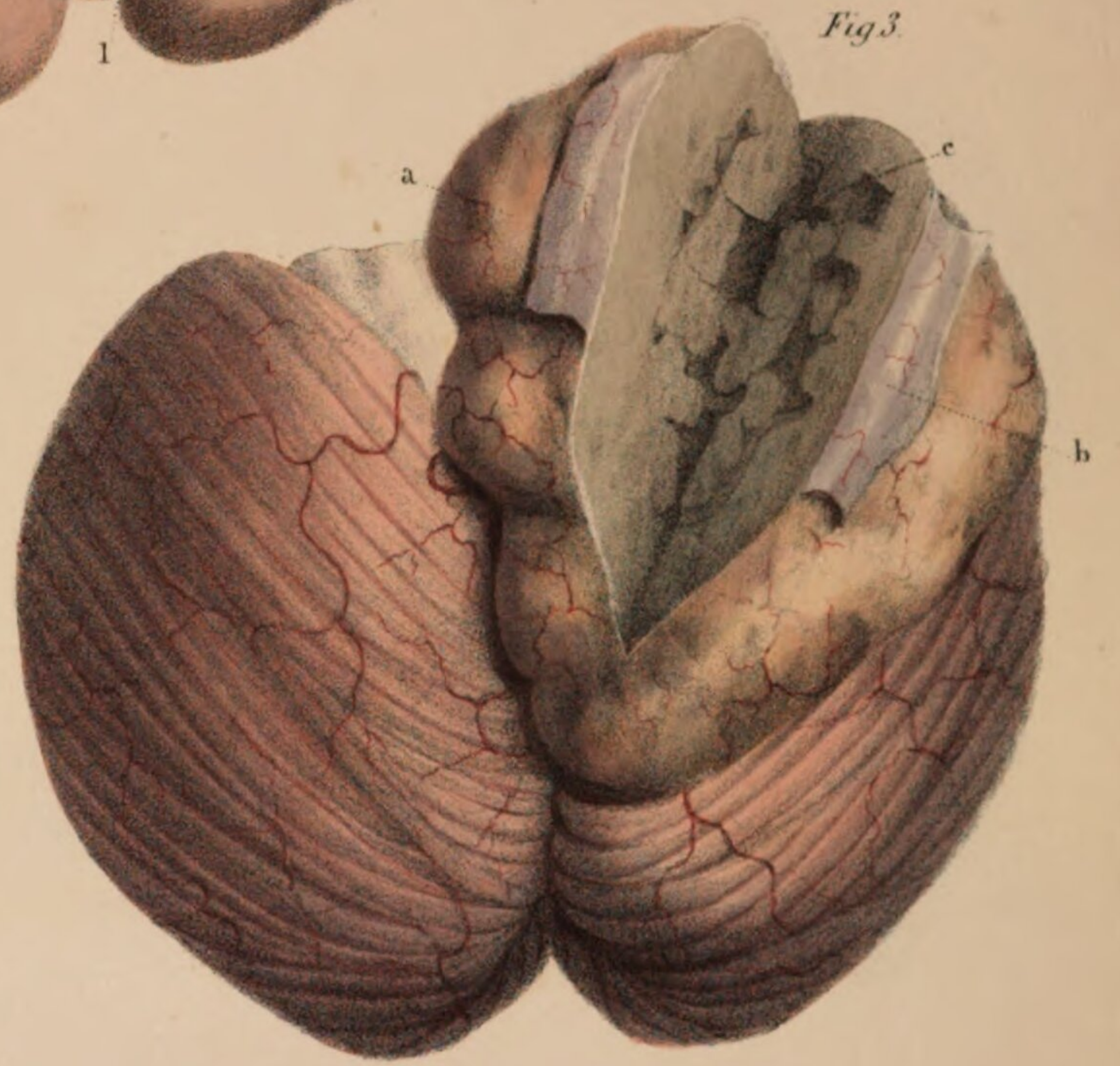
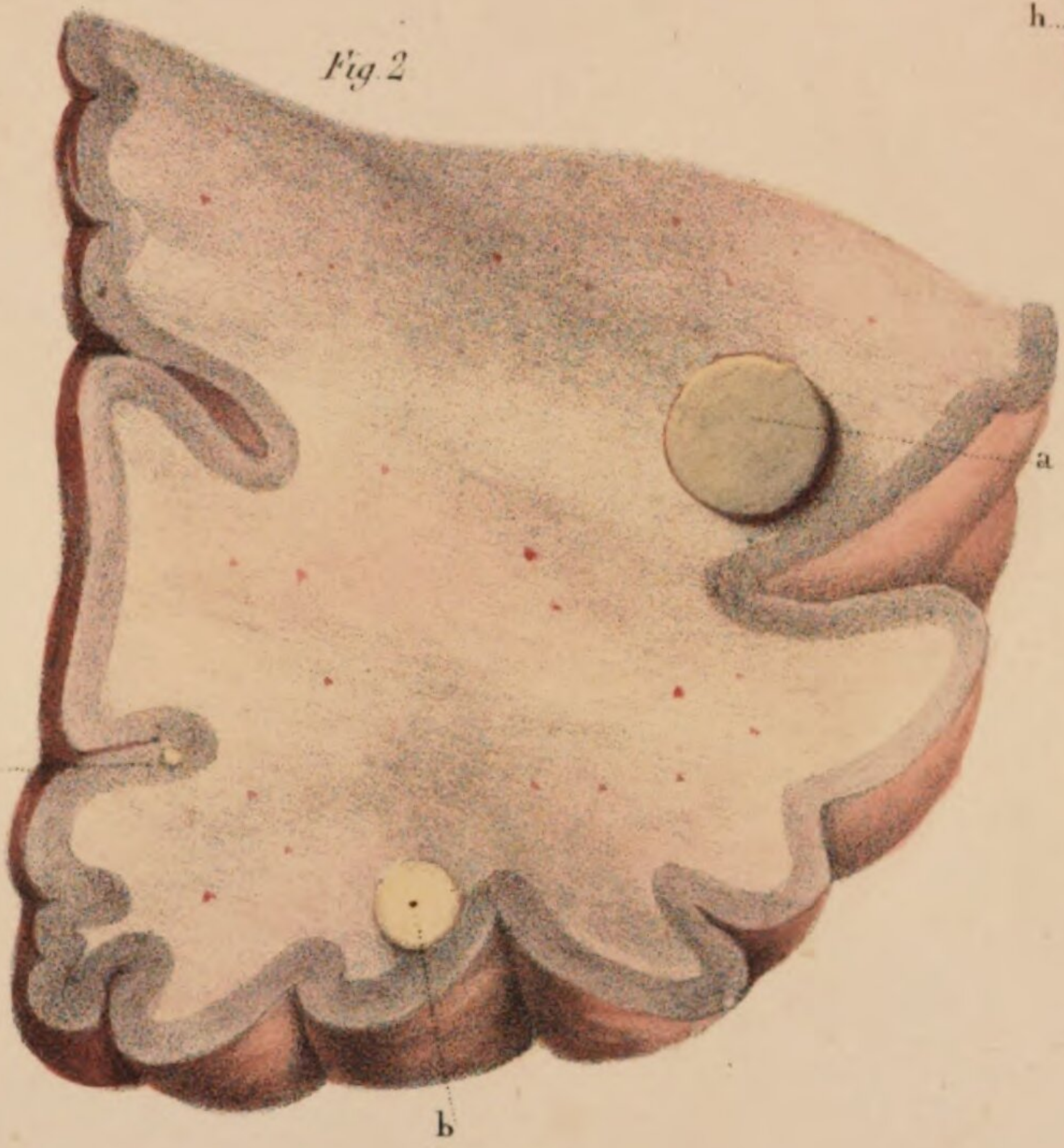


Fig. 6.

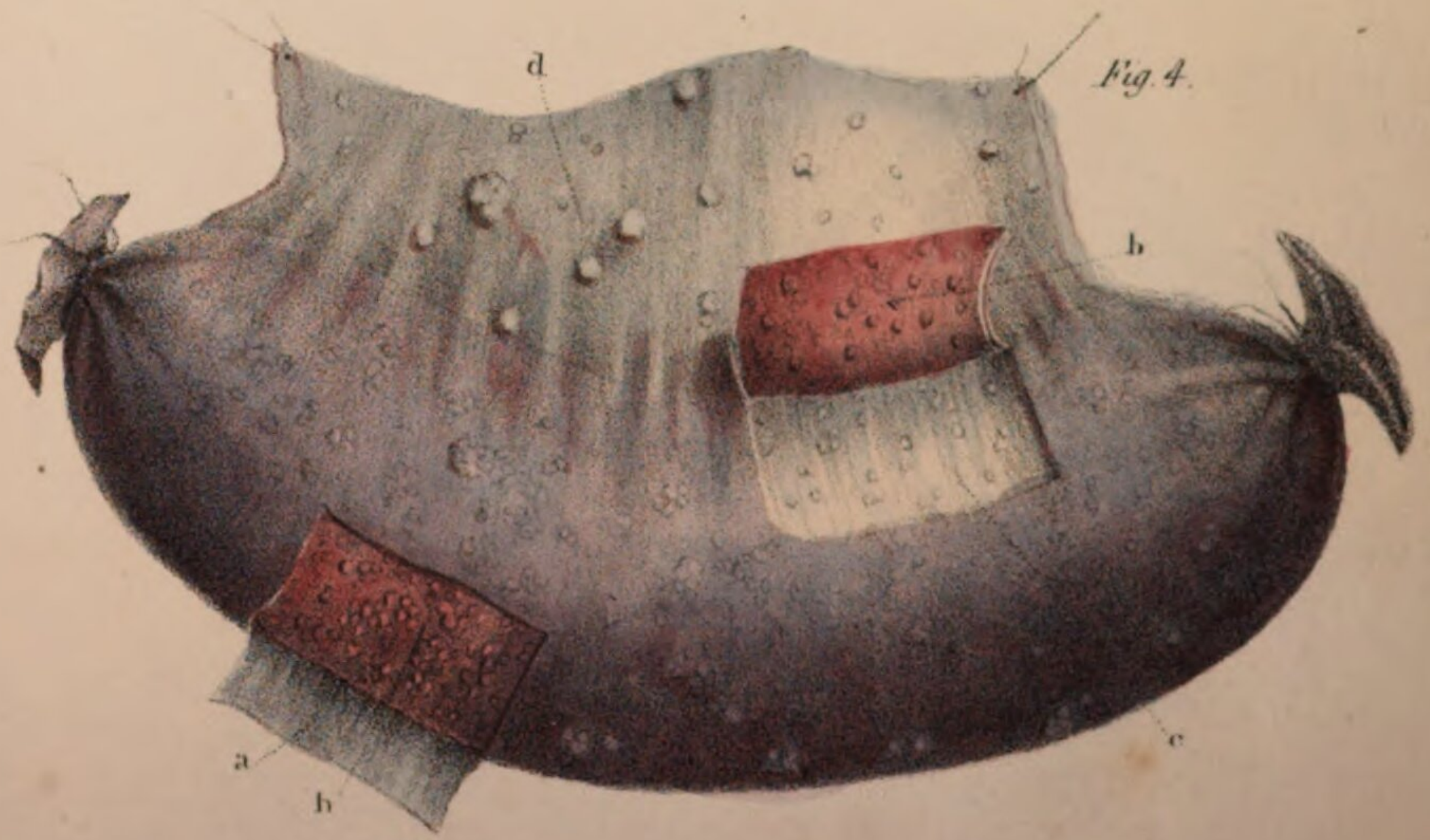
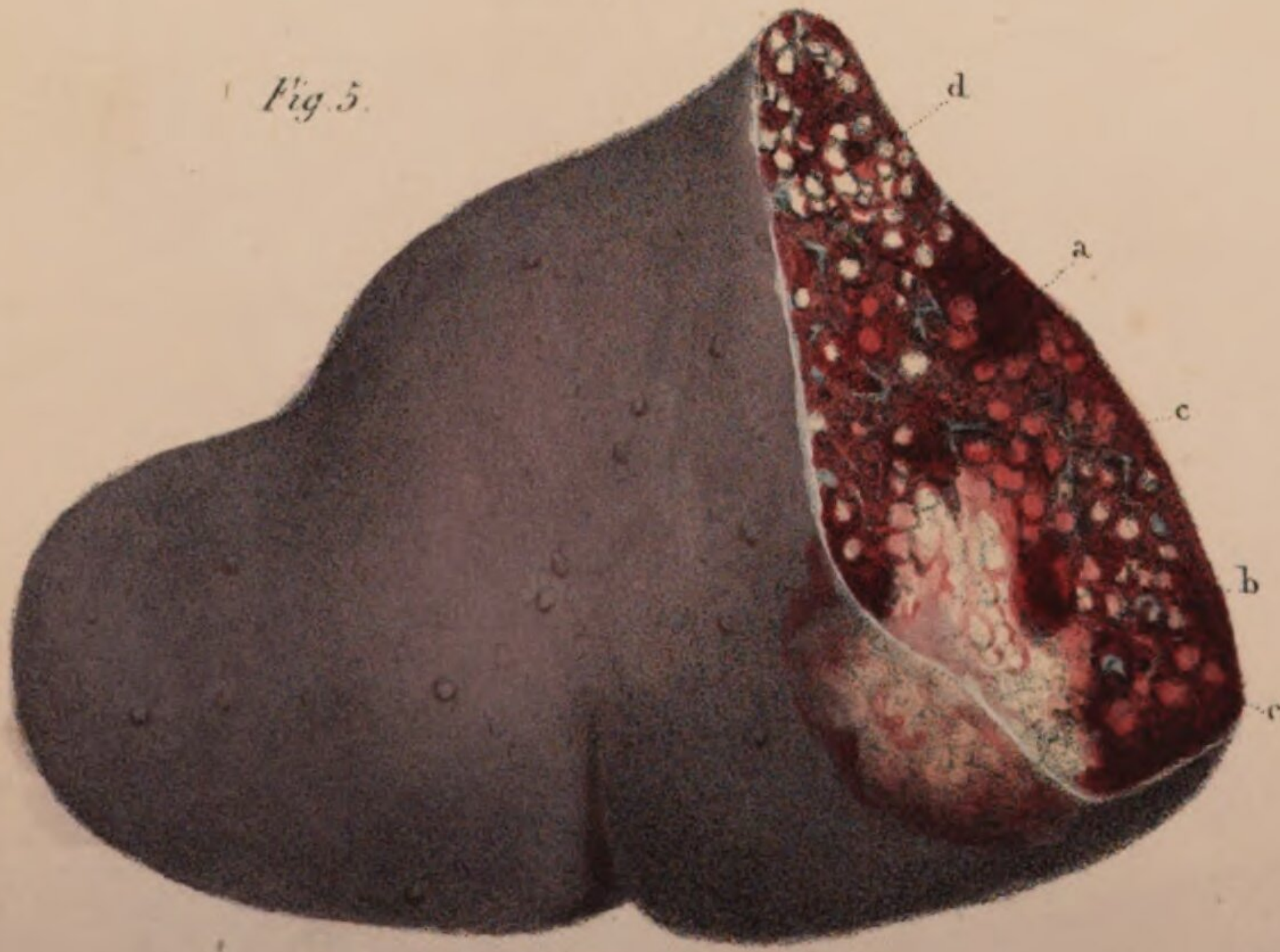


Plate IV.



Carswell, Robert,

Pathological anatomy Illustrations of the elementary forms of disease

London 1838

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